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American Journal of Diseases of Children

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# OBSERVATIONS ON 100 CASES OF THE INFANTILE TYPE OF HEREDITARY SYPHILIS*

BORDEN S. VEEDER, M.D. AND PHILIP C. JEANS, M.D.
ST. LOUIS

As a part of a general investigation of hereditary syphilis, 100 consecutive cases of the infantile type were tabulated for special study. Some points were brought out in this way on which little stress is placed in the usual textbook discussion of hereditary syphilis, and it has seemed of sufficient value to put some of the results of the study on record.

From a clinical standpoint we divide hereditary syphilis into fetal, infantile, late or tardy, and latent types. The term infantile we limit to those cases in which the signs develop after birth and during the first year of life. Although the infection in all types takes place *in utero,* we limit the term fetal syphilis to those infants in whom manifest signs of lues are present at the time of birth; thus it includes abortions, stillbirths, and both premature and full term living infants. Late syphilis was discussed in an earlier paper[1] and we designate as latent, those apparently healthy children of syphilitic parentage who show no symptoms except a positive Wassermann reaction (WR). In this paper we have limited the discussion to 100 consecutive cases of the infantile type observed in the St. Louis Children's Hospital and the Pediatric Clinic of the Washington University Medical School.

An attempt has been made to follow up and keep in touch with every case of syphilis for the past three years, but false addresses, lack of cooperation on the part of parents and the impossibility of making some parents realize that a child in whom all active signs have disappeared is not "cured," have greatly hindered the effectiveness of this work. We have been able, however, to keep in touch for many months with a large part of the cases, and but a few are recorded as having made but one visit to the dispensary. We have even gone so far in some cases as to invoke court aid in forcing parents to have

* Submitted for publication, December, 1915.

* From the Department of Pediatrics, Washington University Medical School, and the St. Louis Children's Hospital.

1. Veeder and Jeans: The Diagnosis and Treatment of Late Hereditary Syphilis, Am. Jour. Dis. Child., 1914, xii, 873.

a syphilitic child treated, although this has usually resulted in the treatment being outlined and supervised by other clinics or private physicians.

### SYMPTOMATOLOGY

In taking histories certain symptoms were carefully inquired into, and these were again noted in the physical examination. As the cutaneous eruptions and the rhinitis or coryza are the most striking lesions objectively, and as they furnish as a rule the complaint for which the infant is brought to the physician, they are the most important of the symptoms. Epiphysitis—or more correctly "Parrot's pseudoparalysis"—while less common, always serves as a chief complaint when present. The spleen is the most important guide to visceral lesions, as it is not normally palpable. An enlargement of the liver is more difficult to classify as an abnormal physical sign, although histologic changes are more common in this organ. The state of the infant's nutrition is also an important factor—to some extent in the symptomatology but more so from the standpoint of prognosis. These symptoms, together with the time of appearance of the first manifestations of the luetic infection, have been considered in detail.

*Cutaneous Lesions.*—A syphilitic rash or a cutaneous lesion of some type was present in 77 cases and absent in 23. Although a number of types of eruption are described by dermatologists as occurring in infants with hereditary syphilis, the cases in our series fell almost entirely into two simple types which were occasionally combined. We have considered in this respect but 64 cases in which the skin lesion was actually seen by us. A macular, or slightly maculopapular rash occurred in 14 cases, while in 48 the eruption was confined largely to an exfoliative or desquamative dermatitis involving the soles of the feet and the palms of the hands. The skin beneath the scaling epiderm is glistening and shiny. We have never seen this type of lesion in an infant unassociated with syphilis, and in our experience it is the most characteristic form the hereditary syphilitic eruption assumes, although not generally so considered. Certain individual cases showed some interesting features in regard to the skin lesion. One infant was brought to the clinic when 3 months old with a rhinitis, at which time the WR was positive. After a few weeks the parents refused to continue treatment and at 10 months the child was again brought for treatment because of the development of a maculopapular syphilid. In this case there was an interval of seven months between the development of the rhinitis and the rash. In three cases the cutaneous lesion was in the form of condylomata. These developed late—tenth to twelfth month—and in two of the cases there had been no earlier skin

involvement, although in the third case there was a history of an eruption at 2 months. Another infant, admitted to the hospital for malnutrition, was found to have ulcers of the groin. The WR was positive and the lesions cleared rapidly under treatment (neosalvarsan). Vulvar ulcerations were noted in one infant a month old. At times small bullae or blebs were present, especially about the moist surfaces, or there was a slight secondary infection, but neither a true bullous dermatitis or pemphigoid eruption, nor a typical pustular syphilid was observed in any of the cases in our series.

*Rhinitis.*—This was present to a greater or less degree in 74 cases and was noted as absent in 26—an incidence almost the same as that of the rash, although they by no means were always associated in the same case. Thus a 9-weeks'-old infant admitted to the hospital for malnutrition developed a syphilitic rash while under direct observation, but at no time was a rhinitis present. The rhinitis was more apt to be present in the cases developing early in infancy and likewise was one of the first symptoms to disappear under treatment.

*Spleen.*—In 82 cases the spleen was definitely palpable while in 18 it could not be felt on physical examination. These negative cases occurred in both the young and older infants. In 3 cases—all over 8 months—an enlarged spleen, together with wasting were the only signs of syphilis present in addition to a positive WR. These are borderline cases between the clinical division into latent and infantile syphilis. We feel it is better to recognize malnutrition as a symptom of the infantile type of hereditary syphilis and limit the term latent to apparently healthy infants and children. In our clinic an enlarged spleen in an infant under 6 months without symptoms of an exudative diathesis has usually proved to be indicative of syphilis or tuberculosis, and hence a WR test is made in addition to the routine tuberculin test which each infant receives. The rachitic spleen is later in development and other causes, such as occasional malarial spleen or a splenic tumor, are uncommon.

*Epiphysitis.*—This was present in 14 cases and absent in 86. In 8 of the 14 cases the epiphysitis developed between the fourth and sixth week. The earliest the lesion was noted was at 2 weeks and in two cases it did not develop until the third month. What we mean by epiphysitis in this connection is really the so-called "Parrot's pseudoparalysis" of hereditary syphilis. Necropsy examination as well as roentgenograms show an epiphyseal lesion to be very common in hereditary syphilis. It is only when the epiphysitis extends to and involves the periosteum and muscles that a lesion which is recognizable clinically is produced, and which is commonly spoken of as epiphysitis.

*Nutrition.*—It is generally accepted that syphilitic infection acts in two ways on the infant. First, by producing specific lesions such as those described above, and secondly, by acting deleteriously on the nutrition and development of the infant as a whole. This malnutrition has been very striking in connection with the infants in our series, and in order to show this we have averaged the weights of the infants at the time of the first examination, as the weight is the most satisfactory index of the condition of nutrition. Of 77 infants whose weight was recorded in the histories, 7 were overweight, 10 of average weight and 60 below average weight. The extremes were from 1,430 gm. overweight, to 3,100 gm. under weight. The 77 infants averaged 1,480 gm., or little more than 3 pounds below the average weight of a like number of infants of the same ages. This condition of malnutrition is not only one of the most constant and striking symptoms of the luetic infection, but it is also one of the most important factors in the production of the high mortality rate of hereditary syphilis, which is discussed below with the question of prognosis.

### TIME DEVELOPMENT OF THE EARLIEST SYMPTOMS

In Table 1 we have recorded the time of the appearance of the first manifestations of the luetic infection as they appeared in ninety-five of the cases. In a few cases the facts were too indefinite to be included.

TABLE 1.—TIME OF DEVELOPMENT OF EARLIEST SYMPTOMS

| Time of Appearance | Cutaneous Eruptions | Rhinitis | Epiphysitis | Wasting and Wassermann Reaction | Jaundice | Hemorrhage and Edema | Total | |
|---|---|---|---|---|---|---|---|---|
| 1 week | 4 | 12 | .. | .. | .. | .. | 16 | |
| 2 weeks | 2 | 3 | .. | .. | .. | .. | 5 | Total in first month, 51 |
| 3 weeks | 3 | 6 | 5 | .. | .. | .. | 14 | |
| 4 weeks | 7 | 6 | 2 | .. | .. | 1 | 16 | |
| 5 weeks | .. | 1 | 1 | .. | .. | .. | 2 | |
| 6 weeks | 6 | 7 | .. | .. | .. | .. | 13 | |
| 7 weeks | 2 | 3 | .. | .. | .. | .. | 5 | Total in second month, 30 |
| 8 weeks | 6 | 1 | 1 | 1 | 1 | .. | 10 | |
| 3 months | 5 | 1 | .. | .. | .. | .. | 6 | |
| 4 months | 1 | 1 | .. | 3 | .. | Iritis 1 | 6 | |
| 6 months | 1 | .. | .. | .. | .. | .. | 1 | |
| 10 months | 1 | .. | .. | .. | .. | .. | 1 | |
| Total | 38 | 41 | 9 | 4 | 1 | 2 | 95 | |

It will be noted that 81, or 85 per cent., of the infants developed symptoms in the first two months of life and about two thirds of these within the first month. The development of symptoms was delayed until late in infancy in a few cases. Thus, one infant first examined at the age of one month and subsequently under constant supervision did not develop signs of lues until the fourth month, when a typical cutaneous eruption appeared and the WR was positive. A rhinitis was the most frequent "earliest symptom" to develop and this was especially so in young infants. Although the rash at times appeared early, there was a tendency for it to be later in development than the rhinitis, in so far as first symptoms are concerned. The other primary symptoms are of too infrequent occurrence to be discussed in detail.

### WASSERMANN REACTIONS

The Wassermann reaction was made in 82 cases. The results were as follows:

| | | |
|---|---|---|
| Positive | 76 or 92.6 | per cent. |
| Doubtful | 1 or 1.3 | per cent. |
| Negative | 5 or 6.1 | per cent. |

In eighteen cases no test was made for various reasons—usually because of very small babies with active signs whose mothers gave a + WR. The percentage of positive reactions, 92.6, is about the same as that usually quoted for the incidence of a positive WR in cases of hereditary syphilis. In an earlier study of the "late" type we found a positive WR in all of a series of 73 cases, or an incidence of 100 per cent. Including these two series we have treated some 300 children with hereditary syphilis in the past 3½ years and in nearly 98 per cent. of these the WR has been positive. One of us[2] found that in a series of 86 mothers of syphilitic children, 73, or 85 per cent., had a + WR—a figure close to that for the + WR in these infantile cases.

It has been found that occasionally very young infants with clinically undoubted syphilis fail to react positively to the WR. Most of these negative findings have been in the first few weeks of life. In this connection our five negative cases are interesting as they do not all come under this class. The data for each is briefly as follows:

1. Treated at 3 weeks. Clinically positive; syphilis in both parents; died before a second test could be made.
2. Tested at 3 weeks. Typical eruption; positive Wassermann in rest of family; given treatment and Wassermann again negative at 8 and 17 months; no late symptoms have developed as yet.
3. Tested at 3 months. Typical eruption and enlarged spleen; Herxheimer reaction following injection of neosalvarsan.
4. Treated in 3 months. Rhinitis; typical eruption; enlarged spleen; Wassermann in both parents positive.
5. Treated at 5 months. Rhinitis; typical eruption; enlarged spleen.

2. Jeans, P. C.: Familial Syphilis, Am. Jour. Dis. Child., 1916, xi, 11.

The last three cases can hardly be placed in the above mentioned well recognized group of clinical infantile syphilis giving a negative WR. It would hardly seem that the usual explanation for these negative reactions—infection late in pregnancy with lack of sufficient time for the formation of antibodies—can be offered for the last three cases, and we can only say that for some unexplained reason there was a failure in these cases of the formation of reactive bodies. None of the three infants had received treatment before the WR was tested. We must recognize that while a negative WR is extremely rare in the late type of syphilis (we have met with but one questionable case), it occasionally occurs in the infantile type.

### PROGNOSIS

The prognosis of an hereditary syphilitic infection is bad. In another paper one of us[2] has shown that of 331 pregnancies occurring in 100 syphilitic families, 131 terminated in death before term, and of the 200 living births, 51 children had died. Another study[3] bearing on this fact compared the total pregnancies occurring in 100 families with contagious disease, 100 taken at random from our records and 83 families in which there was syphilis. The total waste as obtained by taking the total number of living children in relation to the total pregnancies was 25 per cent. for the first two groups and 46 per cent. for the syphilitic group.

It is extremely difficult to get exact figures for the death rate in a series of consecutive cases such as form the basis of this paper, when the infants have to be followed for a long period of time and where the condition under discussion is not acute. Even in countries in which the registration of the population is rigidly enforced this is not an easy task. Thus Welde[4] in Berlin lost track of 32 of 100 polyclinic cases. With the aid of an active social service department and a rather laborious explanation to the parents of each child of what the disease portended, we have been able to keep in touch with nearly all of our infantile cases for periods of many months. The deaths, as will be seen from the figures given below, practically all occur in the first few months and as a general rule infants over 10 months do not die of the syphilis itself. We have found it necessary, therefore, to divide our cases from the standpoint of mortality into three groups:

1. Known dead..... 28
2. Alive and over 10 months of age. (Some now over 3 years.).. 28
3. Infants under 10 months, when last seen..... 44

3. Jeans and Butler: Hereditary Syphilis as a Social Problem, Am. Jour. Dis. Child., 1914, viii, 327.
4. Welde: Ergeb. d inn. Med. u. Kinderh., 1914, xiii, 405.

As to this last group of 44 cases, twenty were lost track of at some period between the second and tenth month and twenty-four are now under observation. Some are in wretched condition and the prognosis is absolutely hopeless; likewise some of those lost from view undoubtedly died. We can confidently say that in at least twelve of these cases the prognosis offers no chance for life, and this figure will more likely reach 15. If these 12 are added to the 28 known deaths in the series the mortality figure for the series reaches 40 per cent., and this despite the fact that many of these infants have been treated intensively with both neosalvarsan and mercury. This figure is but little less than that of Heine,[5] who reported 45 deaths in a series of 100 syphilitic infants in Berlin. In Table 2 the age of the infant at the time of death is shown for the 28 cases.

TABLE 2.—Age of Infant at Time of Death in Twenty-Eight Cases

| Age, Months | No. of Deaths | Age, Months | No. of Deaths |
|---|---|---|---|
| 1 | 4 | 6 | 3 |
| 2 | 5 | 7 | 1 |
| 3 | 4 | 8 | 1 |
| 4 | 4 | 9 | 1 |
| 5 | 4 | 10 | 1 |

It will be noted that the deaths are almost entirely confined to the first half year of life.

It has been surprising to us to find that treatment, *per se*, seems to have but little effect on the immediate outcome of the case. This was a clinical impression which we have found supported by our statistics. We have seen case after case cleared of all syphilitic manifestations as a result of treatment, gradually waste away and die of malnutrition. Even at necropsy no distinctive syphilitic changes have been found in some of these cases and the tissues have been negative when examined for spirochetes. Again, other infants with little or no treatment develop into robust and apparently healthy children. The one factor of greatest importance is the condition of the infant's nutrition. As we have pointed out above, this is impaired in such a degree of frequency and to such an extent that the malnutrition may be regarded as a symptom. As nutrition is to such a large extent dependent on the diet, we have tabulated the relationship existing between the infant's diet and the mortality. We have classified 87 cases, from the standpoint of the food

5. Heine: Prognose u. Symptomatologie d. heredeterie Lues im Saüglingsalter. Jahrb. f. Kinderh., 1910, lxxii, 328.

the infant received into three groups (Table 3). In 13 the record gave insufficient data as regards the diet.

TABLE 3.—Relationship Between Diet and Mortality

| | Living | Dead | Bad Prognosis |
|---|---|---|---|
| 1. Artificially fed, including babies fed less than 1 month on breast | 9 | 17 | 3 |
| 2. Breast fed up to between 1 and 3 months, then artificially fed | 10 | 6 | 2 |
| 3. Breast fed over 3 months (many of these on breast 6 months) | 42 | 3 | 5 |

These figures show clearly the value of breast feeding. The increased death rate of the artificially fed infants with syphilis over that for the breast fed is much higher than the increased rate for artificially fed infants in general. These figures show strikingly what clinical observation has indicated, that the outcome of a case of infantile hereditary syphilis is to a large extent dependent on the condition of the infant's nutrition. While the character of the specific treatment may be of considerable importance in relation to the development of late lesions, the prevention of cerebrospinal syphilis, etc., it is by no means so important as the character of the nutrition as regards immediate life or death. Not infrequently we have seen severe syphilitic lesions disappear with very little treatment and infants in whom symptoms had persisted for a long time make a splendid and rapid recovery, while, on the other hand, infants with what was apparently a mild degree of infection, as manifested by the symptoms, have succumbed after a vigorous and intensive course of treatment. In our experience the prognosis is chiefly dependent on the state of nutrition, and this in the ultimate analysis leads back largely to the manner in which the infant has been fed.

## TREATMENT

It is rather difficult to summarize the results of treatment, yet, on the other hand, it is obviously impossible to discuss a large number of individual cases. We have attempted to carry out various methods of treatment and combinations of drugs in a comparative way, being governed to a large extent in methods by the case as an individual problem. In some cases in our series the duration of the treatment was too short to be of any value in forming an opinion as to the efficacy of the method, and in 11 cases no treatment was instituted for various reasons. We have been able, however, to accumulate sufficient data in 73 cases, in 41 of which neosalvarsan was used in combination

with mercury, and in 31 some form, or forms, of mercury alone was given. As experience has shown that neosalvarsan alone is not as efficacious as when combined with mercury, but one infant was treated in this way. We do not use iodids in the treatment of hereditary lues. In the light of the specific action of mercury and arsenic on the comparatively recently-discovered etiologic spirochete of syphilis, it is hardly a rational procedure, and, moreover, it is a drug not easily borne by the infant. The various combinations and the number of cases in which each was used is shown in Table 4.

TABLE 4.—Form of Treatment and Number of Cases

| | | |
|---|---|---|
| Neosalvarsan | 1 | |
| Neosalvarsan with gray powder | 21 | |
| Neosalvarsan with inunction | 8 | 41 |
| Neosalvarsan, gray powder and inunction | 12 | |
| Inunctions | 13 | |
| Inunctions and gray powder | 7 | 31 |
| Gray powder | 11 | |

The neosalvarsan injections were all given intravenously by means of a glass syringe into the median or external jugular veins, as a rule. The dosage used was 0.075 to 0.15 gm., according to the size of the infant. No bad results were experienced following the injections. The 42 infants receiving neosalvarsan were injected as follows:

21 cases received 1 injection
12 cases received 2 injections
7 cases received 3 injections
2 cases received 4 injections

The deaths were divided proportionately between the cases receiving mercury alone and mercury in combination with salvarsan. For the reasons discussed above, the mortality is of little service as an index of the value of specific treatment in these cases, and we can only form an opinion by considering the effects of treatment on the symptoms.

Neosalvarsan causes a rapid disappearance of the syphilitic lesions, especially of the cutaneous symptoms and the rhinitis. It is more rapid in its effects than inunctions of mercury, and much more so than gray powder used alone. Intramuscular injections of mercury are not suitable for use in infants or in children. It is a simple procedure to inject neosalvarsan intravenously and we use it freely in the outpatient dispensary without admitting cases to the hospital. The only precaution to be taken is that it must be injected directly into the vein without any of the solution escaping into the perivascular tissues. This requires some degree of skill and practice for small veins and hence the external jugular is easier for routine work. In a number of cases the use of neosalvarsan has caused such a rapid disappearance of the rash and rhinitis that it has been difficult to persuade the parents that the condition was not trivial and that the child required further treatment.

The hardest problem as regards the medicinal treatment of hereditary syphilis consists in persuading the parents to persevere in treatment after the active signs of the infection have disappeared. On the other hand, inunctions are unpleasant and very much objected to by the infant's caretaker as a rule. Nevertheless the treatment should be started by one of these two intensive methods of combating the infection. We find it most satisfactory soon to discontinue the one used and change to gray powder, which is the most useful form of mercury we have found for treatment over long periods of time, as it causes as a rule the least disturbance to digestion. If started early, before all active signs have disappeared under the more intensive inunctions or salvarsan therapy, it is more apt to be continued for a longer period of time. The ideal method for practical purposes, and the method which gives best results, in our experience, is two injections of neosalvarsan with a three-day interval followed by a series of courses of gray powder (gr. 1.5 t.i.d.) of a month or six weeks' duration, with an interval of from ten days to two weeks between courses. These should be continued well into the second year of life or until the WR has been made persistently negative, if the case can be kept under control for such a length of time. If neosalvarsan is not available, a course of inunctions of mercury (gr. 10 to 15 daily) lasting two weeks, may be substituted. It is at times surprising to find how the WR persists after seemingly intensive treatment in many cases. We are trying to ascertain how many of these infantile cases in which the extent of the treatment is known develop "late" lesions in childhood, as we know of no studies in regard to this point, but the nature of the material at our disposal makes such a study difficult. A careful control of the diet and of the general hygiene is as essential as the specific therapy as far as the immediate treatment of the case is concerned.

### TREATMENT AND THE WASSERMANN REACTION

In a previous paper we made the statement that we had been unable to obtain a persistently negative WR in a child with "late" hereditary syphilis, despite the most intensive treatment. Although some of the cases reported at that time have been under treatment for a year and a half since, we have no cases as yet which lead us to modify this statement. This, however, does not hold true for the infantile type, as in a number of the cases in this series the WR has become negative as the result of the treatment outlined above and has remained so for many months, nor have these cases developed any further symptoms of lesions of a syphilitic nature. In other words, it seems probable that some cases have been cured, if, as is held at present, a persistently negative WR constitutes a proof of cure. In the majority of

cases, however, it would seem as if the condition had only become latent or quiescent as the result of treatment and only the subsequent course of the case can be determined whether this treatment—which has caused the disappearance of all clinical manifestations, but which has not affected the WR — has been sufficient. From our knowledge of the early treatment of many children with late lesions we are not very optimistic in regard to this group of infants. Cases of apparent clinical similarity respond differently to the same kind and amount of treatment. In some of our cases the WR has become negative, while in others treated in the same way it has remained positive. No general rules, therefore, can be deduced from our cases in regard to the length of time treatment is necessary for the production of a negative WR. Each case in this respect must be treated individually and we can only say that if the development of late lesions is to be inhibited, it is apparently necessary to continue the treatment of an infant with syphilis until the WR has become negative and remained so after an interval of freedom from specific medication.

St. Louis Children's Hospital.

# LOBAR PNEUMONIA IN CHILDHOOD; ROENTGEN-RAY FINDINGS AND AN EXPLANATION OF BRONCHIAL SIGNS.*

HOWARD H. MASON, M.D.
NEW YORK

During the past two years all cases of lobar pneumonia on the Children's Service at the Presbyterian Hospital have been roentgenographed at least once. In those cases showing a shadow, but in which bronchial voice and breathing were not heard, repeated roentgenograms were taken at varying intervals. The purpose of these examinations was to ascertain whether the consolidation as evidenced by this shadow passes through any regular development, and if so, whether the different stages of its progress can be correlated with the development of certain physical signs.

Most textbooks have little to say regarding the site of the early consolidation in lobar pneumonia, or the way it spreads, nor do the books on physical diagnosis correlate the development of the physical signs with the conditions in the chest. When the signs which are considered diagnostic of consolidation appear late or are entirely absent, we find the term "central pneumonia" introduced to explain the unusual situation. Tripier,[1] however, states definitely that the consolidation always touches the periphery of the lung and that the process is most intense at this point. The consolidated area, he says, is usually conical in shape, with its base in the plane of the lung's periphery and its apex pointing toward the center of the affected lobe.

Weill and Mouriquand,[2] from a study of 350 cases by Roentgen ray, conclude that the early shadow is usually triangular in shape, with its base always cortical and generally axillary. They have never seen a shadow that could be considered entirely central. They further state that the shadow is often present long before the signs of consolidation appear and that in some cases these signs never appear. They suggest that the nonappearance of the characteristic signs in these cases may be due to immobilization of the part or to the fact that the base of the shadow lies beneath the scapulohumeral joint.

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* Submitted for publication Jan. 6, 1916.
* Read at the joint meeting of the Philadelphia Pediatric Society, Section on Pediatrics of the New York Academy of Medicine, and the New England Pediatric Society, Philadelphia, Nov. 9, 1915.

1. Tripier: Traité d'Anatomie Path. Gen., Paris, 1904, p. 213.
2. Weill and Mouriquand: Ann. d. méd. et chir. inf., Paris, xvii, 275.

In this study, in order to simplify the problem, all cases complicated by the presence of fluid in the pleura have been excluded. This leaves thirty-seven cases of uncomplicated lobar pneumonia. These all showed definite shadows, which were always quite dense and uniform, and usually definitely outlined. In every case the shadow was so situated

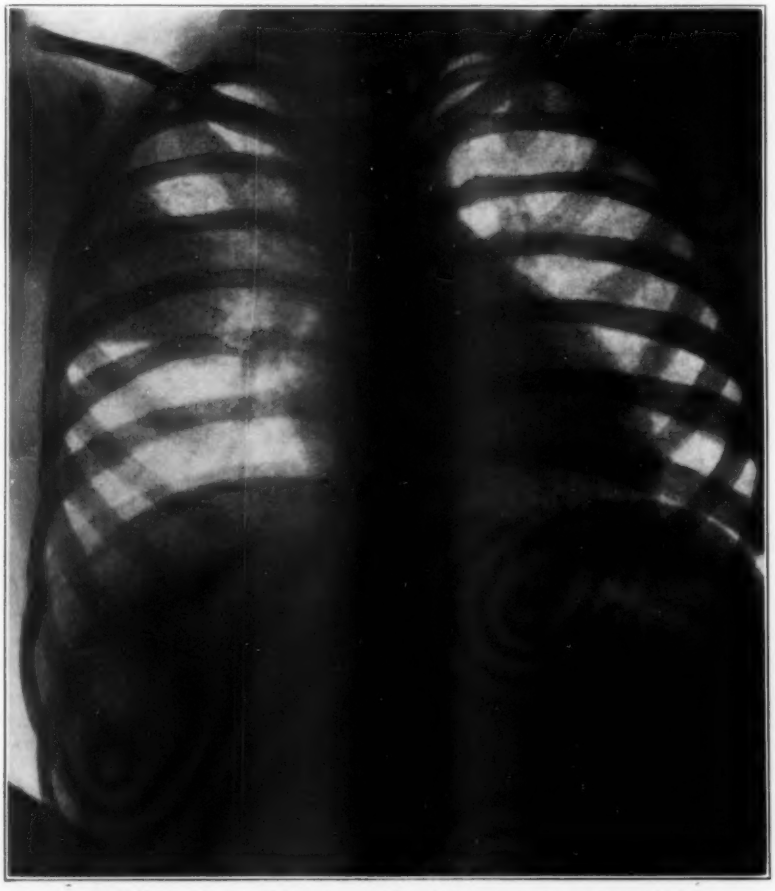

Fig. 1.—(10975) M. S., fifth day. No dulness and no bronchial voice or breathing. Some of these exposures were made in the prone position and some in the supine. This reverses the shadow in some prints.

that the consolidation must touch the pleura at some point. One patient, however, did not show a shadow five hours after the initial chill. A second negative was taken on the third day of his illness and showed a typical dense shadow. Two patients who developed lobar pneumonia while in the ward, were roentgenographed on the second day of fever and both showed definite shadows at this time.

Throughout this article the term "median" has been used to designate shadows which touch the mediastinal shadow in the roentgenograms, but do not extend to the lateral margin of the lung shadow, and the term "lateral" for those shadows that touch the remaining periphery of the lung but do not extend to the mediastinal shadow. Neither of the above terms has been used for a shadow that covers an entire lobe.

As all of the patients were young children, it was found unsatisfactory to take stereoscopic roentgenograms. Accordingly, all

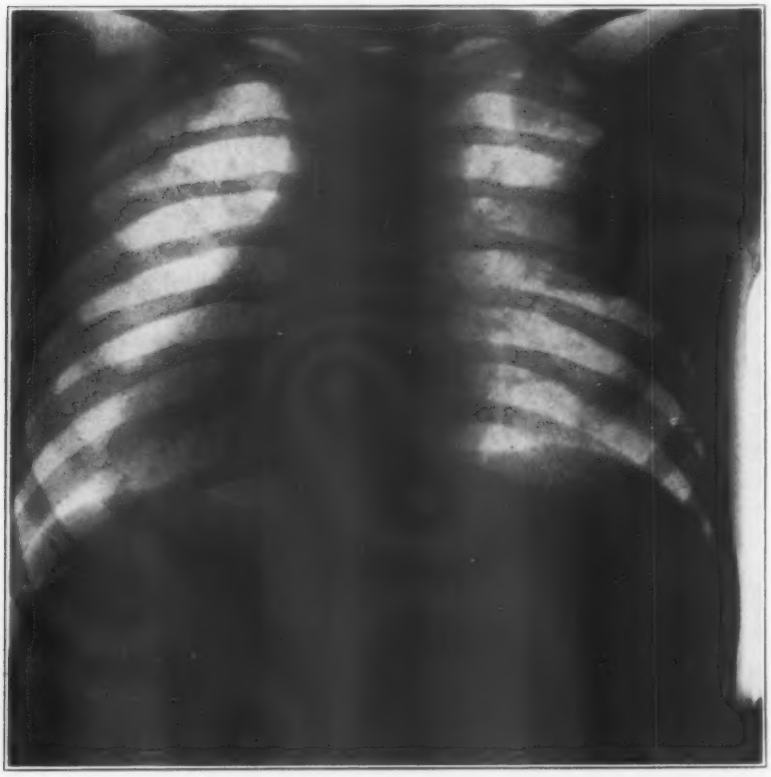

Fig. 2—(10975) M. S., sixth day. Relative dulness and diminished breathing in right axilla. No bronchial voice or breathing.

exposures were made with the child lying on either his back or his abdomen. This makes it impossible to say whether in those cases with median shadows there was normal air-containing lung between the consolidation and the chest wall and between the consolidation and the region of the root or not. In the cases with lateral shadows, the greater part of the shadow always lay close to the pleura, while the depth to which the shadow extended varied in different cases and at different

stages of the same case. Without stereoscopic negatives it is impossible to say definitely from a study of the roentgenograms that the more median portions of such shadows were not due to surface consolidation also. Physical signs, however, helped to settle this point. In the cases with lateral shadows, the early dulness was present over the base of the triangle; that is, over the lateral part of the shadow and not over the more median portion of the shadow as it should have been if this part of the consolidation were close to the chest wall.

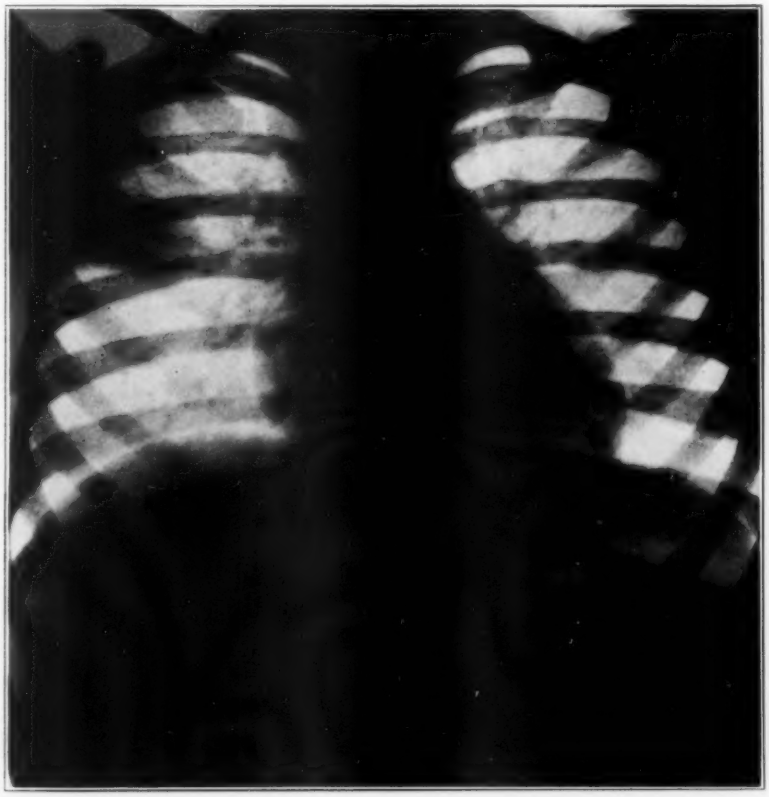

Fig. 3.—(10975) M. S., eighth day. Signs same as on sixth day, with a few moist râles.

Of these thirty-seven cases, fifteen showed in the first roentgenogram a uniformly dense shadow extending over all or almost all of at least one lobe, thus making it impossible to tell where the shadow first appeared. The first negatives of seven cases showed median shadows. In these cases also it was impossible to say how deep the consolidation extended. In both groups the shadow touched the pleura at some point. In thirteen cases the shadows were lateral, and it is the further

development of these shadows that I wish to consider. At first they were always triangular in shape, with the base on some portion of the periphery of the lung and the apex directed towards the root of the lung. The most frequent site of the base of the triangle was opposite the upper part of the axilla. From day to day they usually spread upward and downward and towards the root, gradually increasing until just before the crisis. Occasionally they spread on the surface

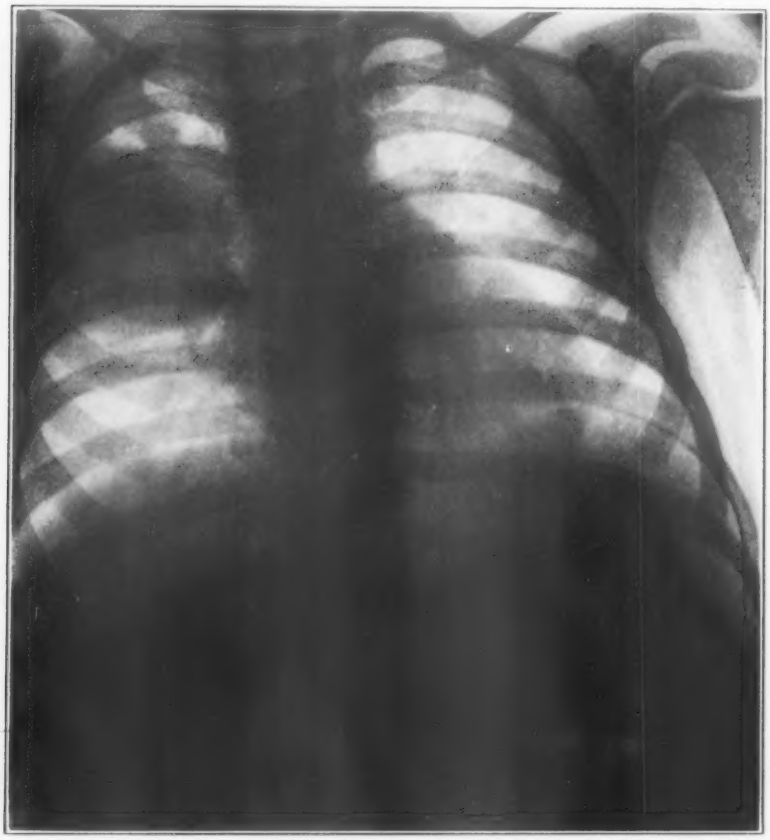

Fig. 4.—(12187) E. S., sixth day. Dulness but no bronchial breathing or voice.

of the lung but not towards the root. In the early stages there was a relatively clear area between the apex of the shadow and the root of the lung. As the shadow spread it obliterated this clear area, until in most cases there was a uniform dense shadow extending from the lateral surface of the lung to the root.

That is to say, in the beginning of the process there is a triangularly shaped shadow with its base on some portion of the lung periphery

and its apex pointing toward the hilum. This shadow is separated from the root by normal lung shadow. Gradually the triangular shadow spreads up and down and towards the root into the normal area until, as a rule, most of one lobe is involved. In trying to visualize the nature of the anatomic change in the lung, Tripier's description helps us. At first there is a small conically shaped consolidation, the base of the cone lying in the plane of the lung periphery and the apex of the cone pointing toward the hilum. This cone of consolidation gradually

Fig. 5.—(12187) E. S., seventh day. Dulness and bronchial breathing and voice.

becomes larger and larger until finally its apex reaches the hilum and its base coincides with all, or with a large part, of the pleural surface of the lobe. The process seems, therefore, to begin in that portion of the lung which is anatomically farthest from the hilum, that is to say, that part of the lung which lies close to the pleura. To be sure the early consolidation may lie along the mediastinal pleura, or the diaphragmatic pleura or the pleura of an incisure. The fact which I desire to emphasize is that the consolidation always begins in that portion of the lung

which lies close to the pleura. Such a consolidation, when situated on the mediastinal or diaphragmatic surface of the lung may be separated from the chest wall by normal air-containing lung and in this sense may be central. On the other hand, this conception is not the same as that usually understood when so-called "central pneumonia" is spoken of, the term central pneumonia being in this sense restricted to consolidation which does not touch the pleura at any point. Weill and

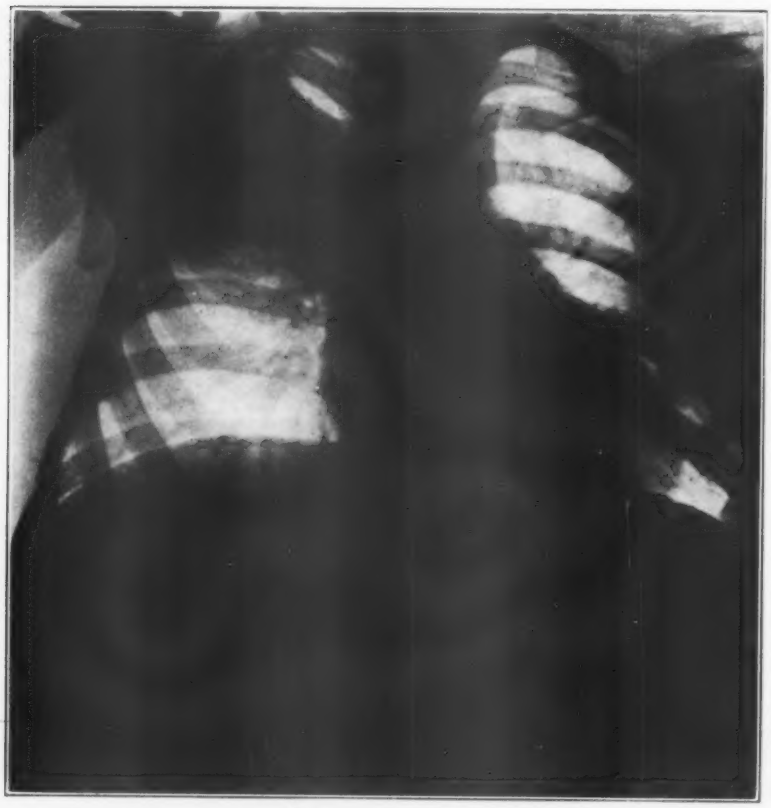

Fig. 6 (12187) E. S., eighth day. Dulness and bronchial breathing and voice.

Mouriquand agree with this point of view when they say that they have never seen a shadow that could be interpreted as due to a central penumonia.

The shadows which I have described and the anatomic changes which they indicate, suggest the explanation for the occurrence of certain physical signs with which we are familiar.

In all of the fifteen cases that gave dense shadows extending from the lateral surface of the lung to the region of the root in their first

roentgenograms, there were bronchial voice and breath sounds. Thirteen cases showed only lateral shadows at the first examination and in none of these were bronchial voice and breath sounds detected. Ten of these patients were examined again later when additional roentgenograms were made. Eight of the ten developed bronchial breath and voice sounds later. In all eight cases these signs did not appear until the shadow extended to the region of the root. The shadow in the two cases that did not develop bronchial signs did not extend to the hilum, but the shadow in these cases was always separated from the root by an area of normal lung shadow. Unfortunately no subsequent roentgenograms were made in the remaining three cases.

In all these cases bronchial voice and breath sounds were present when the shadow extended from the lateral surface of the lung to the region of the root, and they were absent when the shadow was separated from the region of the root by an area of normal lung shadow.

Nine cases are not included in the above. Seven of them showed median shadows in their first roentgenograms. Three of the seven gave bronchial signs at the time the first negatives were taken, while the remaining four all developed bronchial signs later. The other two cases had shadows on the left side, the median extent of the shadows being obscured by the cardiac shadow. In these nine cases, because of their position, it was impossible to determine whether there was continuous consolidation from the chest wall to the region of the root.

There can be little doubt that those cases with continuous shadows from the lateral surface of the lung to the region of the root, have corresponding areas of consolidation. These facts furnish an explanation for the presence of bronchial signs. Tyndal has shown that sound is conducted much better through a medium of uniform density than through one of varying density. Bronchial breath and voice sounds are normally present over the trachea and large bronchi, but they are not conducted to the rest of the chest. In the first case, the sounds traverse solid tissue of a comparatively uniform density with little loss in quality or change in intensity, while in the latter case they pass through many layers of tissue and air of altering density, the repeated changes from air to tissue being accompanied by loss of the original quality.

When the medium is uniform, therefore, there is bronchial breathing and voice. If, however, the consolidated lung extends only part way from the stethoscope to the hilum, the loss in quality is great and the breath and voice sounds are not bronchial.

Thus, in the fifteen cases with shadows from the lateral surface of the lung to the root, the situation of the shadows suggested that the consolidation was so placed as to cause bronchial breathing and voice

over the consolidated area. In the thirteen cases where the shadows were merely lateral at first, the bronchial breath and voice sounds did not appear until the shadows and the consolidation bridged the area of normal lung to the root and supplied a medium of uniform density.

The same explanation can be used to account for the presence or absence of bronchial breath and voice sounds in cases with pleural effusion. If the lung beneath the fluid is airless, due to any cause (compression, consolidation, tumor, etc.), the sound traverses a medium of comparatively uniform density and suffers little loss. In these cases the voice and breath sounds over the fluid are bronchial. If the lung beneath the fluid contains air, the loss of sound is great and bronchial voice and breath sounds are not present. Montgomery and Engelhardt[3] have come to the same conclusion.

## SUMMARY

Thirty-seven cases of lobar pneumonia were studied with the Roentgen rays, most of them repeatedly. All thirty-seven cases showed a definite shadow.

The shadow was always so placed that it touched the pleura at some point.

The early shadows were triangular in shape, with their bases on the pleura and their apices separated from the region of the root by normal lung.

In their later development the shadows extended in size and became uniform from the periphery to the root of the lung. When the shadow involved this entire stretch, bronchial voice and breathing were present, but not otherwise. It is believed that the inference is justified that the dimensions of the shadows correspond to the extension of consolidation.

## CONCLUSIONS

The following conclusions may, therefore, be drawn:

1. The consolidation of lobar pneumonia in children begins in that portion of the lung which lies just beneath the pleura.

2. A central pneumonia in the strict sense never occurs. Silent consolidations are subpleural consolidations and are separated from the hilum by normal lung.

3. Bronchial breath and voice sounds are dependent on the presence of a medium of comparatively uniform density from the site of their origin (the trachea and large bronchi) to the point where the ear or

3. Montgomery and Engelhardt: Arch. Int. Med., 1915, xv, 1040.

stethoscope is applied. These conditions are fulfilled when the consolidated area extends from just beneath the ear to the region of the hilum.

### PROTOCOLS OF CASES WHOSE ROENTGENOGRAMS ARE SHOWN IN THE ILLUSTRATIONS

No. 10975. M. S., girl, aged 4½ years. Admitted on the fourth day of illness. Crisis on the sixth day. Leukocytes fifth day, 48,000; polynuclears, 88 per cent. Fifth day, no dulness and no bronchial voice or breathing. Roentgenogram shows right lateral shadow extending a little more than half the distance to the root. Sixth day, day of crisis, relative dulness and diminished breathing in right axilla. No bronchial voice or breathing. Roentgenogram shows slightly less extensive shadow. Eighth day, two days after crisis, relative dulness and diminished breathing in right axilla with a few moist râles. No bronchial voice or breathing. Roentgenogram shows a less extensive shadow.

No. 12187. E. S., girl, aged 1 year. Admitted on fifth day of illness. Crisis on ninth day. Leukocytes fifth day, 33,600; polynuclears, 78 per cent. Sixth day, dulness but no bronchial breathing or voice over right upper. Roentgenogram shows extensive shadow over most of right upper, but separated from region of root by a comparatively clear area. Seventh day, dulness and bronchial breathing and voice over right upper lobe. Roentgenogram shows slightly more extensive shadow with clear area near root almost obliterated. Eighth day, dulness and bronchial breathing and voice over right upper lobe. Roentgenogram shows shadow slightly denser near root.

I wish to thank Mr. Ira L. Simms, radiographer, and Mr. C. J. Lander, photographer, for their painstaking care, without which this work would have been impossible.

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# RECURRENT HILUS INFILTRATION

## AN UNUSUAL FORM OF TUBERCULOSIS IN CHILDREN *

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Our knowledge of the pathogenesis and the anatomic varieties of tuberculosis in childhood has undergone considerable change during the past decade, and at present we know that the clinical types of pulmonary tuberculosis in the first few years of life are quite distinct from those of adults. Typical apical lesions commonly found in adults are not seen till late in childhood, while affections of the bronchial lymph nodes are the rule. There are, however, forms of tuberculosis whose localization in the lungs of children is just as typical as that in adults, the area of lung involved being determined by the presence of the diseased bronchial nodes. It is our purpose in this paper to report clinically and from the roentgenographic standpoint one particular variety of juvenile tuberculosis which is of considerable importance.

Before describing the lesion in detail it may be well to review briefly the generally accepted conception of the origin and distribution of tuberculosis in the infant and child. The work of Hamburger, Ghon, Albrecht and others has proved that in the majority of instances tuberculosis enters the organism by means of the respiratory tract and first localizes in a small, often almost microscopic, lesion in the lung tissue itself. This focus, varying in size from that of a pinhead to an almond, is termed by Ghon the primary focus. This has been found in practically all the cases giving a positive von Pirquet reaction. Secondarily from this lesion the bronchial nodes become involved and form in all probability the nidus in which the latent infection lies dormant. The disease may remain permanently in this condition, the nodes finally organizing or calcifying; or else they may act as a starting point for further pulmonary involvement. This secondary involvement may be the outcome of either a reinfection, in which case the apical and phthisical forms result, or else direct extension from the lymph nodes may take place.

The form of tuberculosis here to be described is one of such extension from the lymph nodes and occurs frequently enough to warrant

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* From the Pediatric Dispensary and the Roentgen-Ray Department of Mount Sinai Hospital.

its incorporation as a clinical entity in the great group of tuberculous infections of the lung. Sluka[1] has encountered it thirty-eight times in a few years, and Eisler[2] has also reported an identical clinical picture. For a knowledge of its morphology we are indebted entirely to the Roentgen ray, which discloses it with striking clearness.

This lesion occurs in the vast majority of cases on the right side and its point of predilection is the lung in the immediate vicinity of the fissure between the upper and middle lobes. Beginning with its base at the hilus, where a dark shadow of tuberculous nodes is visible, a fairly opaque shadow of homogeneous density extends outward into the lung for a considerable distance, gradually fading into the normal

Fig. 1.—(Case 1). Radiograph of chest taken March 6, 1915. Enlarged bronchial lymph nodes surrounded by triangular infiltration at the root of the right lung. Thickened interlobar fissure indicated by arrow.

lung tissue. The resulting infiltration is triangular in shape, its apex near the axilla. Its lower margin is sharply limited by the interlobar fissure. The latter is evidently thickened and is the seat of a pleurisy,

1. Sluka, E.: Ueber Roentgenbefunde bei Tuberkulosen Kindern mit Expiratorischem Keuchen, Wien. klin. Wchnschr., 1910, xxiii. Die Hilustuberkulose des Kindes im Roentgenbilde, Wien. klin. Wchnschr., 1912, No. 7. Ein Weiterer Beitrag zur Hilustuberkulose des Kindes im Roentgenbilde, Wien. klin. Wchnschr., 1913, No. 7.

2. Eisler, F.: Die Interlobare pleuritische Schwarte der Kindlichen Lunge im Roentgenbilde, München. Med. Wchnschr., 1912, No. 35.

as it can usually be seen extending quite to the axilla beyond the limits of the infiltrated area.

Various interpretations have been applied to these shadows. They have been assumed by Eisler[2] to be to a great extent due to pleurisy in and about the interlobar fissure, resulting from a primary tuberculous focus in the sense of Ghon, which is so frequently found at necropsy in this situation. Sluka, on the other hand, and with greater plausibility, believes that it is a direct infection of the pulmonary tissue from the caseated lymph nodes at the hilus with a resulting tuberculous infiltration of the lung. This is rendered more likely by the following con-

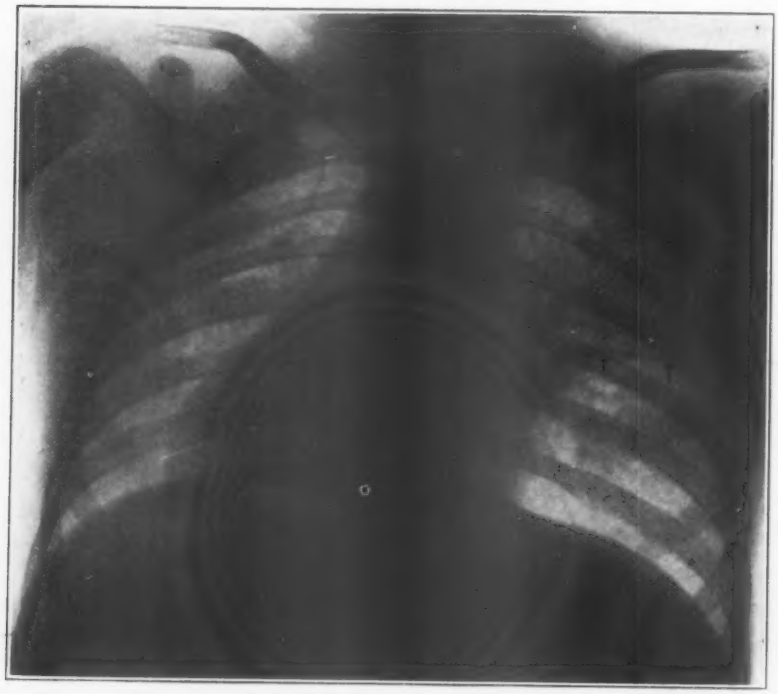

Fig. 2.—(Case 1). Radiograph taken March 25, 1915. Absence of hilus infiltration. Persistence of thickened interlobar fissure. Enlarged bronchial lymph nodes.

siderations: The primary lesion of Ghon is commonest in the first years of life. If it were responsible for this form of tuberculosis, we should encounter it frequently at an early age. It is, therefore, remarkable that Sluka in his series never found it before the age of 2 years, and we have had a similar experience in the Roentgen examination of children.

The infiltration does not remain constant in size or appearance, and this is perhaps the most remarkable fact in regard to these cases.

Most forms of tuberculosis as we encounter them in adults, are characterized by the constancy of the findings over fairly long periods of time. In these cases, the process suffers striking variations in its extent. The Roentgen examination made at intervals of a month may show at first a wide area of infiltration and at the later examination, nothing may be left but the thickened interlobar fissure. The latter usually persists for a long time as a thin, sharp line running horizontally across the upper part of the right lung from the inner end of the fourth costosternal junction to the axilla. Such a sequence of infiltrated and normal lung may be repeated several times, and justifies the appellation of "Recurring hilus tuberculosis," and will find its correspondence in the signs and symptoms of the patient.

Fig. 3.—(Case 1). Radiograph taken May 8, 1915. Recurrence of hilus infiltration as in Figure 1, sharply limited below by the interlobar fissure.

Tuberculosis of the bronchial lymph nodes may therefore give rise to two distinct pathologic changes in their immediate vicinity, which may occur separately or coincidently. The first is the form of infiltration described in this paper. The second is an interlobar pleuritis which frequently accompanies the infiltration and is well shown in the illustrations. This pleuritis may, moreover, occur alone and give rise to

a similar group of symptoms. Roentgen-ray examination is therefore absolutely necessary to differentiate between the two conditions.

Children in whom this typical hilus involvement has been found are usually brought to the physician on account of general debility, loss of weight, slight cough or night sweats. In childhood the condition is Physical examination of the chest is usually completely negative unless found between the ages of 2 and 8 years, not occurring in infancy. the process has involved a considerable mass of lung tissue. This is a point of the greatest importance for it emphasizes the fact that in

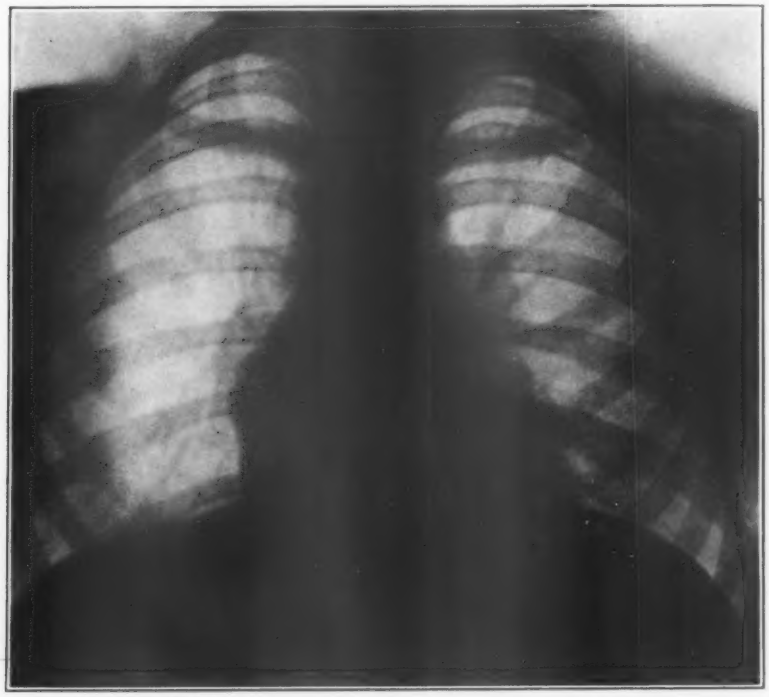

Fig. 4.—(Case 1). Radiograph taken November, 1915. Absence of all infiltration.

such debiliated children with positive von Pirquet test, Roentgen-ray examination is imperative. This is all the more necessary since it has been shown that this particular lesion is in many cases a curable one, provided appropriate treatment is followed. When the lesion is extensive, slight dulness to the right of the sternum may be present, and expiration may be prolonged, harsh and accompanied by a few fine râles. These signs though important when present, must not be relied on as we have seen involvement of considerable extent where no signs whatever were apparent.

The clinical picture of these cases is, moreover, peculiar, in that it shows considerable variation in the same case at different times. There is apparently a recurrent inflammatory process, which as it extends from the hilus into the lungs produces an exacerbation of the clinical signs and symptoms. At such times there is more rapid emaciation, more weakness, more fever and possibly more cough. It is during such exacerbations that the Roentgen ray reveals the extension into the lung fields as described above. In favorable cases, and the majority seem to be of this type, there is a distinct regression, the symptoms all becom-

Fig. 5.—(Case 2). Radiograph taken June 3, 1915. Enlarged bronchial lymph nodes surrounding right lower bronchus. Triangular shadow of infiltration in right upper lobe bounded below by the interlobar fissure.

ing much milder, and the Roentgen-ray coincidently showing a diminution in the amount of lung involvement. Sluka has followed many such cases through a number of similar cycles.

## REPORT OF CASES

The following cases are chosen from a number observed as typical of the lesion under discussion:

CASE 1.—Sarah D., first seen November, 1914, at the age of 3 years. Family history negative; no exposure to tuberculosis. The child had always been well

till three months before coming under our observation, when she had an attack of measles, followed by "pneumonia." Since that time the child has had a cough and has not appeared to be in good health.

*November 21, 1914.* Child rather poorly nourished. Except for slight general glandular enlargement, physical examination negative. Pirquet strongly positive. Weight 35 pounds.

*December 17, 1914.* Still coughing and running very slight temperature. A few râles to be heard in the right chest posteriorly at the angle of the scapula. A roentgenogram taken at this time shows enlarged bronchial lymph nodes and a linear shadow of a thickened right interlobar fissure.

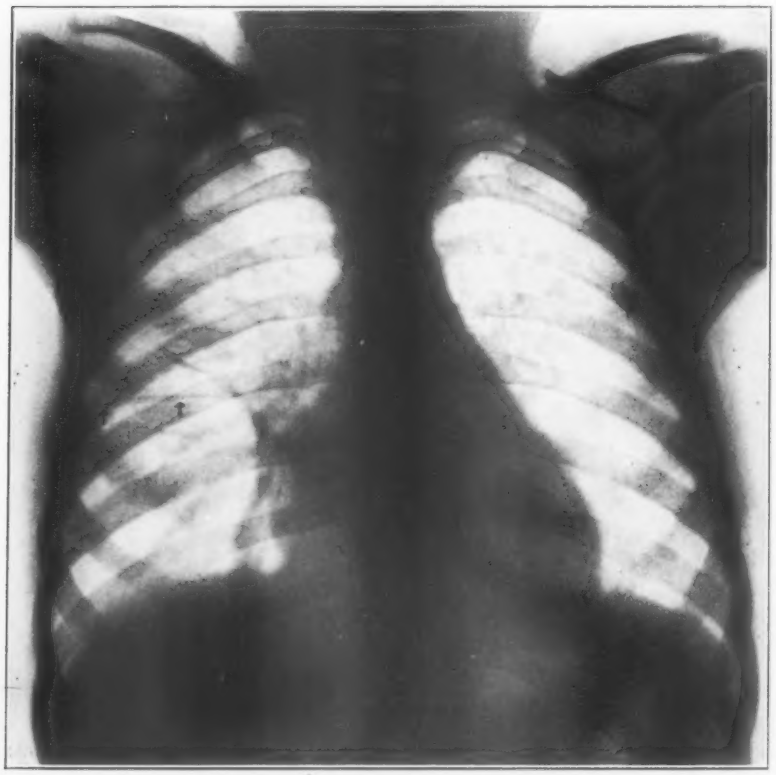

Fig. 6.—(Case 2). Radiograph taken June 29, 1915. Disappearance of the infiltration present in Figure 5. Note the persistence of the interlobar fissure which is well shown throughout its entire extent.

*January 16, 1915.* Cough persists.

*March 6, 1915.* Recently child has begun to complain of dull pain in the right side of chest. There is slight dulness and bronchovesicular breathing over the middle portion of the right chest posteriorly. The Roentgen-ray shows distinct consolidation in the region of the interlobar fissure between the right upper and middle lobes. (Fig. 1.)

*March 25, 1915.* Feeling much improved. Cough improved, but still present. Gained in weight. Physical signs now show only somewhat roughened breath-

ing over the middle lobe. Roentgenogram (Fig. 2) shows a resorption of the process, leaving only a thickened interlobar fissure.

*May 8, 1915.* After an interval of about six weeks during which the child was in apparently good health, the symptoms again recurred. Physical signs same as during last attack. Roentgenogram (Fig. 3) shows a recurrence of the infiltration, which has the same distribution as at the previous exacerbation, but is more extensive.

*November, 1915.* After the last attack the child was sent to the mountains where she improved rapidly. Has been well since then. No coughing. No

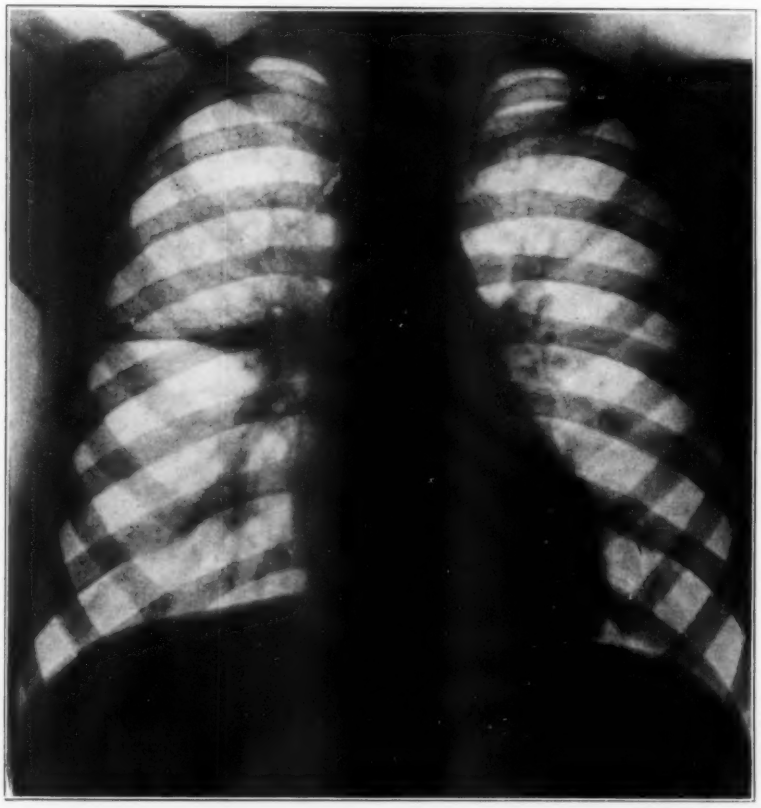

Fig. 7.—(Case 3). Interlobar pleurisy. Dense shadow along the interlobar fissure. Note the width of the shadow peripherally, as shown by the arrow.

fever. Physical condition is now excellent. Has gained rapidly in weight; weight now 46½ pounds. Color good. Nutrition excellent. Examination of lungs reveals no abnormality whatever. The Roentgen-ray examination shows enlargement of the bronchial nodes, but no infiltration of the lung. (Fig. 4.)

CASE 2.—Minnie C.[3] Aged 6 years. History negative except that three years prior to this report she had an acute pulmonary condition accompanied

3. From the dispensary service of Dr. M. Taschman, through whose courtesy we report this case.

by cough, fever and vomiting. Since then she has had recurrences of such attacks every few months. In the interval she is apparently well.

*June 3, 1915.* Fairly well nourished child, small for her age. The only physical finding worthy of note is dulness in the middle of the right chest anteriorly and posteriorly. No changes in breath sounds. Pirquet test positive. Roentgenogram (Fig. 5) shows a triangular shadow at the right hilus, with a marked thickening of the interlobar fissure.

*June 29, 1915.* The child is apparently well again and has neither symptoms nor physical signs. Roentgenogram (Fig. 6) shows a persistence of the interlobar fissure but no pulmonary infiltration.

Pleurisy of the interlobar fissure, which may give rise to symptoms very similar to those found in the cases of hilus infiltration, is well illustrated in Figure 7, the plate of a 6-year-old tuberculous child, who for four years has had recurring attacks of cough, fever and sweats. Here may be noted the exudate along the fissure and infiltration of the hilus. It may be evident that to differentiate this case from those of the other group without a Roentgen examination would be impossible, as in neither case are there any marked physical signs.

The exact pathogenesis of these lesions is rather obscure, for corroboration by necropsy is impossible, since the majority of the children recover, and those in whom the lesion goes on to a fatal termination present the picture of a general caseous involvement of the lung. The histories of many of the cases have shown that before the onset of the condition the child had either measles or pertussis; and the theory has been advanced that possibly there is at this time not only a lowering of the resistance to tuberculosis, but also that local conditions in the bronchial glands may be at fault. It is well known that during the course of measles the nodes become swollen and inflamed, and it is possible that this inflammation added to the tuberculosis already present causes a thinning of their capsules and final rupture either into a bronchus or directly into the surrounding pulmonary tissue.

The fact that such regressions not only in the symptoms, but also in the Roentgen-ray findings, can take place also needs explanation. It is difficult to conceive of a true tuberculous infiltration coming and going in this fashion, and the most plausible theory appears to be that the underlying pathologic process is one of an inflammatory exudate about the tuberculous hilus. Such exudative processes have been described by Fraenkel,[4] who ascribed them to toxin action and showed that they might resolve leaving very few traces behind them.

This condition should not be confused with interlobar pleural exudates such as have been quite recently described by Weihe.[5] These

4. Fraenkel A.: Klinische und anatomische Mittheillungen über indurative Lungenentzundung, Deutsch. Med. Wchnschr., 1895, No. 10.

5. Weihe, F.: Die Interlobare Pleuritis im Kindesalter, Ztschr. f. Kinderh., 1915.

may be differentiated by the fact that they often occur in infants and in nontuberculous individuals (in Weihe's cases seven out of eight). When of considerable size they also produce more distinct clinical signs and their Roentgen-ray shadow has a tendency to appear broader at the periphery than at the hilus, the opposite of the condition as seen in our cases. In doubtful cases exploratory puncture should be performed.

From the clinical aspect, nontuberculous inflammatory processes occurring in children who have pulmonary tuberculosis are also well known, and we may quote the following from Bandelier and Roepke's[6] *"Klinik der Tuberkulose"*:

> Particularly worthy of note in children with bronchial gland tuberculosis is the tendency to certain affections of the bronchi and lungs, which are in no respect tuberculous in their nature. Here belong, according to Hutinel, the disposition to simple diffuse bronchitis, frequent localized bronchitis in the lower lobes, at the apices or in the neighborhood of the hilus, also certain forms of asthmatic bronchitis, and finally pulmonic congestion which may completely simulate pneumonia, but disappear very rapidly, sometimes in the course of a few hours. We are dealing in such cases with reactions on the part of the respiratory tract, or with indirect sequelae of hilus tuberculosis. Their differentiation from focal pulmonic disease is always difficult, often only possible from a consideration of their clinical course. One should therefore be careful about hurried diagnoses and unfavorably given prognoses.

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ADDITIONAL REFERENCES

Rach, E.: Radiologisch erkennbare anatomische Typen der Kindlichen Lungentuberkulose, München. Med. Wchnschr., 1914, No. 8. Zur Klinik der Bronchialdruesentuberkulose im Kindesalter, Beitr. zur Klin. der Tuberc., 1914, xxxii. Beitrage zur Roentgendiagnostik der Lungen tuberkulose im Kindesalter, Ztschr. f. Kinderh., 1913, viii.

6. Bandelier and Roepke: Die Klinik der Tuberkulose, Wuerzburg, 1914.

# MESENTERIUM COMMUNE WITH ABNORMAL COURSE OF JEJUNUM

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Congenital anomalies of the abdominal organs are now treated, not merely from an exclusively anatomic standpoint as curiosities, but have been given especial attention since the development of the embryology and teratology, and particularly since the advance in abdominal surgery. Displacements and malformations of the intestinal tract, in the first place, may have great diagnostic interest and may present problems complicating operative technic. Cases observed clinically or anatomically with exactitude therefore possess a practical interest beside a theoretical value, and the surgeon must ever be prepared to meet such anomalies and deal with those variations of normal abdominal topography depending on them.

As a contribution to this teratologic topic I report the observation of the following case, in which congenital anomalies were detected at the anatomic section of the pediatric course at the Rush Medical College.

Boy, 1 year old, died from "decomposition" and pneumonia, Oct. 14, 1915, in the Cook County Hospital.

On opening the abdomen loops of small intestine present themselves, entirely covering the colon ascendens and transversum, with the exception of the hepatic flexure. On removal of the convolutions of the small intestines the cecum with the appendix appear in the region of the lower pole of the left kidney, and prove to be completely and freely mobile. This position naturally must be considered merely as accidental. The colon ascendens has its course from here upward and to the right to the hepatic region. It is completely free and mobile, without any fixation to the posterior abdominal wall, and has a long mesentery in common with the ileum and transversum, so that the intestines can be drawn far upward and placed on the chest. Two folds pass from the hepatic flexure over the duodenum to the liver and kidney, respectively.

The mesocolon transversum in the region from the duodenum to the spleen is fixed in a normal way. The flexura coli lienalis is well fixed, the spleen itself is possessed of a well-formed saccus lienalis.

The mesocolon descendens, though fused with the posterior abdominal wall, is free in the left flank, so that the colon is mobile and can be lifted up about 1 cm.; the mesentery is more than 2 cm. wide (fresh specimen) at the point where it crosses the left crista ossis ilei, and the sigmoid flexure is very long, forming secondary loops and having an unusually deep recessus subsigmoideus; it can be drawn 2.5 to 3 cm. beyond the right crista ossis ilei.

After lifting up the mesocolon transversum nothing of the duodenojejunal flexure is to be found at the normal site or in its proximity. Only on lifting up the mesentery of the colon ascendens and the adjoining ileum does the

Absent coalescence of colon and mesocolon ascendens with posterior wall of abdomen and with duodenum. Free colon ascendens with appendix and convolutions of ileum and jejunum turned upward. Jejunum emerging on posterior aspect of mesenterium commune from fossa, marked by match.

jejunum become visible; it emerges on the posterior surface of the ileocecal mesenteric plate from a fossa that is formed by a peritoneal fold; after lifting up also the mesentery of the small intestines, as shown in the illustration, the jejunum can be followed up to the duodenal flexure proper. The fold of the fossa starts from the mesocolon near the insertion of the appendix, crosses the jejunum and ends in the mesentery of the latter. This fossa is located below the middle of the length of the mesentery. If the cecum is brought to the normal site, the jejunum descends alongside the vertebral column, and the fossa has its place near the promontory. The part of the jejunum that is enwrapped in the plate is of course freely mobile up to the duodenum.

The radix mesenterii is very narrow and peduncle-like; taking its course with the arteria mesenterica superior, it has a very small insertion on the vertebra near the duodenum; thus the mesentery resembles a fan, the free edge of which is fringed with coils, with the narrow peduncle inserted on the vertebra.

The mesenterium ileocolicum is 5.5 cm. long measured from the ileocecal angle to the fixation of its root (preserved and much shrunk specimen). The transverse diameter of the abdomen is 11.5 cm. between the crista ossis ilei.

The arteria mesenterica superior does not, as normally, cross the horizontal, preaortic part of the duodenum, but within the root of the mesentery it ascends to the left of the duodenum toward the aorta.

The duodenum is not covered by the mesocolon, as the figure shows; its ascending portion is somewhat mobile, being incompletely fused with the peritoneum parietale of the posterior abdominal wall. The pars libera of the omentum majus, spreading over the small intestines, usually more or less apron-like, is undersized, especially in its left part. There is no ligamentum hepatorenale.

On the posterior wall of the abdomen there are nowhere scar formations, bands or adhesions visible; the peritoneum has everywhere its normal luster.

In briefly summarizing, we find the following anomalies present: mesocolon commune; duodenojejunum behind the plate of the mesocolon commune, the jejunum emerging from an abnormal fossa, and running downward along the vertebral column; abnormal course of the arteria mesenterica superior; ascending part of duodenum somewhat mobile; sigmoid flexure elongated with wide range of mobility even considering the age.

## DISCUSSION

In order to understand the principal features of this anomaly, we may briefly recall to mind a few phases of the development of the mesentery and intestinal tube in the human embryo. As known, the omentum majus develops as an outgrowth of the mesogastrium, which is attached to the posterior wall of the abdominal cavity in the region of the pancreas and spleen on the one side, and to the great curvature of the stomach on the other side. By intense proliferation of this peritoneal plate a bag-like duplicature is formed, that covering apron-like the colon and small intestines reaches with its folded edge usually into the small pelvic cavity. An underdevelopment of the free part (bursa omenti) of the omentum is not rare in the adult, all degrees in size being known, from the normal type that reaches with its lower edge to the small pelvis, even to the most rudimentary development with a length of a few centimeters, or even, indeed, absence of this part. In the new-born we often find a less developed pars libera.

For the normal position and arrangement of the intestines and mesentery the process of development and rotation of the free original umbilical loop (*Nabelschleife*) as described by Toldt (1879), is important. Under normal conditions the cecum and colon which develop in the lower, recurrent segment of the *Nabelschleife* move to the right side of the abdominal cavity, the cecum descending finally to the iliac region. The mesentery of the colon ascendens (mesocolon ascendens) coalesces with the primary parietal peritoneum of the posterior abdominal wall, as likewise does the mesocolon descendens, so that only the colon transversum retains its mesentery mobile and with both its surfaces free. The formation of the normal peritoneal covering at the junction of the duodenum and jejunum is due to the rotation of the primitive umbilical loop and the resulting overlying of the root of the mesentery by the colon transversum and its mesocolon, which latter crossing the spinal column to the right side undergoes partial coalescense with the posterior wall of the abdomen.

Anomalies as described above, and similar ones, originate, because under certain conditions the normal coalescence of the mesenteries and intestinal segments with the posterior abdominal wall does not take place. This developmental disturbance affects most often the colon ascendens. In such case it is possessed of a freely mobile mesocolon, which passes without sharp demarcation into the iliac mesentery. Occasionally also the coalescence of the duodenum or of the colon and mesocolon descendens is entirely or partially absent, the segments of the intestines concerned retaining their free and mobile mesentery. The rotation of the primitive umbilical loop may also occur in the reversed direction with "situs inversus intestini" resulting, or it may be more or less incomplete. Cases of failure of rotation have been reported by Charles Mayo,[1] Beekman Delatour,[2] and Downes.[3] The unusually deep recessus subsigmoideus in my case is nothing else than the unusually large residue of the originally entirely open space between the abdominal wall and the mesentery of the sigmoid flexure; the coalescence of the mesosigmoid with the parietal peritoneum being defective. It is evident that extraordinary mobility of the intestines and their mesenteries renders possible abnormal positions of these organs; by coalescence or adhesions, antenatal or postnatal, of the latter at unusual places, abnormal relations are formed, making the anomalies of position of intestines and mesentery finally permanent.

The fusion of primarily free mesenteries, as those of the colon ascendens and descendens, with the parietal peritoneum are secondary physiologic processes.

1. Mayo, Charles H.: Med. Rec., New York, March, 1912, p. 401.
2. Delatour, B. H.: Ann. Surg., 1915, lxi, 73.
3. Downes: Ann. Surg., 1915, lxi, 111.

Why these morphologic changes should be disturbed, resulting in malformations and misplacements, that is, the causal genesis, is not known with certainty. While some authors as Klaatsch and Koch[4] consider the anomaly under discussion, as likewise an excessive growth of the colon, as atavism, others presume that abnormal length of the intestinal tube causes incomplete apposition to and fusion with the parietal peritoneum.

Mazylis[5] describes a case of mesenterium primitivum (commune) in combination with congenital cystic degeneration of the kidneys; he also attributes the lack of the normal coalescence to the physical factor of lack of space within the abdomen due to the enlargement of the kidneys. Though in this case a causal relation between both anomalies may exist, it is interesting to take notice of the fact, emphasized in the doctrine of teratology, that in the presence of one malformation we often may expect in the same individual some other congenital anomaly; often irrelevant, insignificant and without any direct mutual connection.

The clinical significance of the mesenterium commune mobile lies in the fact that it creates a predisposition of the intestines towards twisting around the mesenterial axis and towards displacements, not possible under normal conditions. The persistence of the embryonic stage in the development of the mesentery in addition to failure of coalescence, manifests itself also in the form of its root; while normally the fixation of the radix mesenterii extends from the second lumbar vertebra obliquely towards the right iliac fossa, the radix of the mesenterium commune is usually limited only to the second lumbar vertebra and is therefore very narrow. The disproportion of the width of the loop at the insertion of the intestinal tube and at the peduncle-like radix of the mesentery undoubtedly is a mechanical factor favoring axial twist (volvulus), the more so since the entire intestinal tract inserted on the common mesentery represent so to speak one single loop, hanging on a narrow peduncle. One only needs to load the entire intestinal convolutions on the palm of the hand, holding the common peduncle between the fingers, to realize at once the great mobility, and readiness of torsion of the mesentery. This anomaly may even in the later life of the individual cause serious and even fatal conditions, especially dangerous when not early recognized, and therefore surgeons are especially impressed with the clinical significance of this condition.

The knowledge of such anomalies is important in deciding the difficult problems arising for diagnosis; cases as described above will also

4. Klaatsch and Koch: Quoted from Huebner, Virchows Arch. f. path. Anat., 1910, cci, 427.

5. Mazylis: Reviewed, Monatschr. f. Kinderh., 1913, xii, 178.

make apparent the great technical difficulties to be encountered in an operation for appendicitis, when the diseased organ is buried deeply behind the small intestines at the site of the lower pole of the left kidney. Also the congenital anomaly of the duodenojejunal loop, as represesented in my type, may be of considerable interest for the surgeon, who frequently performs gastrojejunostomy. Boyd[6] during an abdominal operation contemplating gastrojejunostomy encountered a topographic anomaly of the duodenojejunal flexure somewhat simliar to the above case. The coil could not be found at the usual situation after lifting up the transverse colon and mesocolon, but emerged from under the hepatic flexure and passed vertically downwards to the right iliac fossa.

Armstrong[7] described the observation of a duodenum with long mesentery and abnormal position; it ran to the right and turned downwards external to the hepatic flexure, ascending colon and cecum.

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6. Boyd, S.: Brit. Med. Jour., 1914, ii, 507.
7. Armstrong: Tr. Am. Surg. Soc., 1910, xxviii, 299.

# HEMOREFRACTOMETRY IN INFECTIOUS DISEASES OF CHILDREN *

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Refractometry is yet an almost blank chapter in pediatrics. In contrast to the now numerous papers on refractometry in adults, such researches in children are rare.

On normal refractometry of blood serum in healthy children we know only the papers of Reiss[1] and Nast.[2] According to Reiss the refractometric index of blood serum in nurslings is lower than in adults, and this decrease agrees with about a 2 per cent. lower concentration of serum proteins. From the age of 6 to 10 months, says Reiss, the refractometric index rises and attains a definite degree, with a value varying from 1.3480 to 1.3514, as in adults. Refractometry is perhaps the best and surest means of serum protein estimation; Reiss's researches on the subject are too well known to need any detailed mention here.

In order to estimate protein concentration in blood serum, we subtract from the number read 1.33320, which is the value of n/D to distilled water, plus 0.00277, corresponding to the other substances in solution in blood serum, and then divide the remainder of the two successive subtractions by 0.00172, the refractometric variation to 1 per cent. protein solution. So if we find $n/D = 1.34872$ to be the refractometric index of a serum, we have $1.34872 - (1.33320 + 0.00277) = 0.01285$, and $0.01285 : 0.00172 = 7.41$, then the protein concentration of this serum is 7.41 per cent.

According to Nast there are in infancy five periods of protein concentration in blood serum, as follows: from 1 to 3 months; from 3 to 8 months; from 8 to 18 months; from 1½ to 5 years and from 5 to 14 years, with a progressive increase, and is the same as that in adults during the last two periods.

In order to have a paradigm for our next following observations, we have estimated blood serum-protein concentration in healthy infants with Abbe's total refractometer. Reiss's and Nast's studies were made with Pulfrich's immersion refractometer, which gives a number of five decimals, while Abbe's refractometer gives only four.

* Submitted for publication, Nov. 9, 1915.
* Read before the Brazilian Society of Pediatrics, Oct. 10, 1915.

1. Reiss: Jahrb. f. Kinderh., lxx.
2. Nast: Ztschr. f. Kinderh., 1914, p. 92.

My results in contrast to those of Reiss and Nast, are as shown in Table 1.

TABLE. 1.—Comparison of Refractometric Results

| Age, Months | Reiss | | Nast | | Mello-Leitão | |
|---|---|---|---|---|---|---|
| | N/D | Proteins, Per Cent. | N/D | Proteins, Per Cent. | N/D | Proteins, Per Cent. |
| 1— | 1.34557 to 1.34747 | 5.6 to 6.7 | ..... | .... | ..... | .... |
| 1 | ..... to 1.34580 | ... to 5.7 | 1.34503 to 1.34826 | 5.26 to 7.20 | 1.3449 to 1.3465 | 5.19 to 6.12 |
| 2 | ..... to 1.34654 | ... to 6.1 | 1.34605 to 1.34672 | 5.69 to 6.25 | 1.3463 to 1.3468 | 6.0 to 6.29 |
| 3 | 1.34635 to 1.34659 | 6.0 to 6.2 | 1.34523 to 1.34690 | 5.96 to 6.34 | 1.3461 to 1.3468 | 5.88 to 6.29 |
| 4 | 1.34629 to 1.34740 | 6.0 to 6.6 | 1.34540 to 1.34749 | 5.46 to 6.70 | 1.3463 to 1.3472 | 6.0 to 6.52 |
| 5 | 1.34721 to 1.34802 | 6.5 to 7.6 | ..... to ..... | .... to .... | 1.3469 to 1.3472 | 6.36 to 6.52 |
| 6 | ..... to ..... | ... to ... | ..... to ..... | .... to .... | 1.3470 to 1.3475 | 6.41 to 6.58 |
| 7 | ..... to ..... | ... to ... | 1.34694 to ..... | 6.38 to .... | 1.3470 to 1.3474 | 6.41 to 6.52 |
| 8 | ..... to 1.34976 | ... to 8.0 | ..... to 1.34695 | .... to 6.38 | 1.3470 to 1.3478 | 6.41 to 6.87 |
| 9 | ..... to ..... | ... to ... | ..... to 1.34798 | .... to 6.98 | 1.3469 to 1.3478 | 6.35 to 6.87 |
| 10 | ..... to 1.34733 | ... to 6.6 | 1.34884 to 1.34891 | 7.48 to 7.50 | 1.3471 to 1.3480 | 6.47 to 7.0 |
| 11 | ..... | ... | 1.34820 | 7.10 | 1.3480 | 7.0 |
| 12 to 18 | 1.34895 to 1.34920 | 7.5 to 7.7 | 1.34779 to 1.34823 | 6.88 to 7.12 | 1.3480 to 1.3485 | 7.0 to 7.28 |
| 18 to 60 | 1.34894 to 1.35110 | 7.5 to 8.8 | 1.34854 to 1.35006 | 7.31 to 8.18 | 1.3482 to 1.3500 | 7.11 to 8.15 |

The results show infancy to be divided into four periods of albuminemic concentration: Under 3 months, when n/D is below 1.3470; from 3 to 8 months, with n/D varying from 1.3478; from 8 to 18 months with n/D varying from 1.3480 to 1.3485, and over 18 months, with n/D values above 1.3482. Chart 1 shows the refractometric figures of Reiss, Nast and mine.

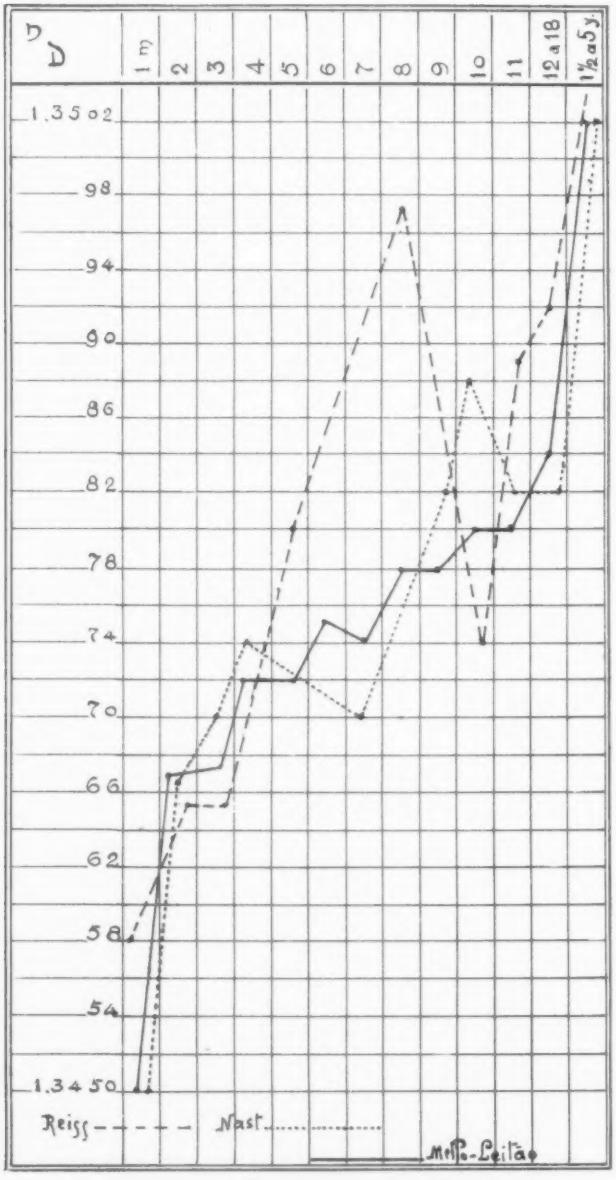


Fig. 1.—Normal refractometric index in infancy.

We have seen that as soon as the child takes table food the refractometric index rises and attains adult values, and this index varies within the same bounds after 18 months.

Refractometry of the blood in infectious diseases has been the subject of papers by Landelowski, Oppenheimer and Reiss, and Achard

and Saint-Girons. Achard, Touraine and Saint-Girons[3] in their researches on rheumatism, pneumonia and typhoid fever have shown a very near relation between sickness evolution and serum protein concentration. These authors consider, in the course of an acute infectious disease, four albuminemic phases as follows:

First phase, the percentage of proteins falls more or less regularly;

Second phase, albuminemy decreases to the lowest degree;

Third phase, protein concentration rises slowly or swiftly; and in the

Fourth phase, exceeds the normal degree, descending to it again little by little.

TABLE 2.—PROTEIN CONCENTRATION IN MEASLES

| Name | Age, Months | Fever Days | Eruption Day | Koplik | N/D | Proteins, Per Cent. |
|---|---|---|---|---|---|---|
| Olivia | 10 | 4 | 2d | + | 1.3452 | 5.36 |
| Heitor | 7 | 3 | 1st | + | 1.3463 | 6.0 |
| Jandyra | 23 | 4 | 1st | + | 1.3471 | 6.52 |
| Nair | 34 | 3 | 1st | + | 1.3470 | 6.41 |
| Octavio | 6 | .. | 3d | — | 1.3448 | 5.12 |
| Dalila | 18 | .. | 4th | — | 1.3470 | 6.41 |
| Christovão | 5 | .. | 12th | — | 1.3466 | 6.17 |
| Luiz | 8 | .. | 25th | — | 1.3480 | 7.0 |
| Heitor | 36 | 3 (39 C.) | 1st | + | 1.3475 | 6.58 |
| | | 5 (37.5 C.) | 3d | + | 1.3463 | 6.0 |
| | | | 8th | — | 1.3485 | 7.86 |
| | | | 15th | — | 1.3503 | 8.33 |

In the first phase the albuminemic percentage is directly proportional to the duration and gravity of the infection; thus, in pneumonics whose disease endures from six to nine days, the least percentage of proteins was 7.224; in typhoidics, whose disease protracts itself to twenty days or more, the percentage descends to 6.771. The decrease is at first abrupt, becoming slower as it approaches the minimum, and while there is fever, the protein percentage progressively descends to the minimum and remains there.

In the second phase the refractometric index comes to minimum when the fever falls and the temperature tends to become normal; in

3. Achard, Touraine and Saint-Girons; Arch. de méd. expér. et d'anat. path., 1912, xxiv, 647.

diseases whose fever falls suddenly, the least degree of protein concentration is attained the day or the afternoon of crisis; in lysis, on the last day, when the fever decreases slowly.

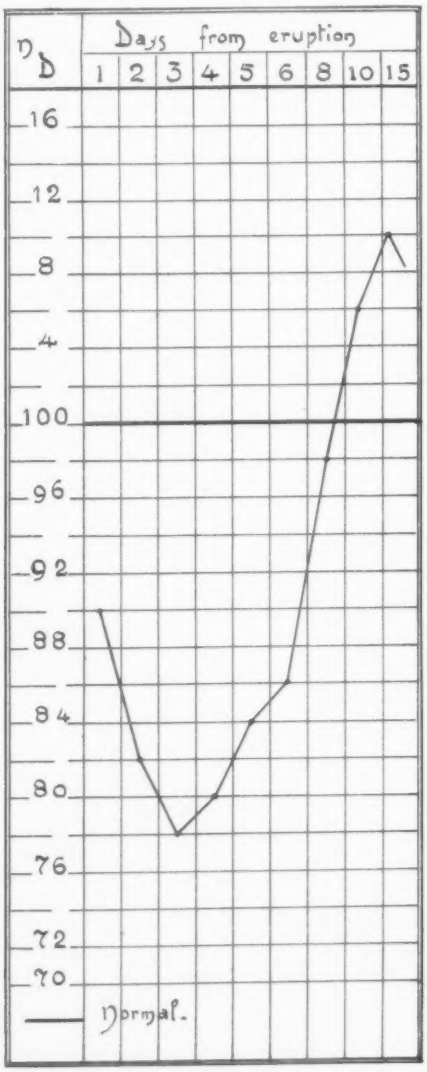


Fig. 2.—Hemorefractometry of measles as compared with the normal.

This second albuminemic phase occurs according to two clinical states. (a) It is only a slight condition, and in figures is represented by an acute angle between the descending line of the first phase and the ascending line of the third; (b) there is a more or less lasting condition,

during which the refractometric degree maintains itself constant, or with very little oscillation; in figures it is represented by a horizontal bar joining the oblique lines of the first and third phases. The first figure is observed when there is crisis, and the second when termination is by lysis.

The protein percentage rises after the day without fever, and in general this ascent is at first slow and afterward more rapid; sometimes the rise is rather sudden (only when there is crisis).

The fourth phase is not constant, but often the protein concentration goes far beyond the normal, and afterwards decreases little by little.

TABLE 3.—Refractometric Observations in Whooping Cough

| Name | Age, Months | N/D | Proteins, Per Cent. | Observations |
|---|---|---|---|---|
| A. M. | 36 | 1.3472 | 6.52 | Nauseous cough since 6 days. |
| C. S. | 4 | 1.3455 | 5.54 | Nauseous cough since 3 days. |
| S. M. | 7 | 1.3490 | 7.57 | Spasmodic cough since 1 month. |
| S. S. | 10 | 1.3510 | 8.73 | Spasmodic cough since 50 days. |
| L. F. | 11 | 1.3480 | 7.00 | Spasmodic cough since 75 days. |
| G. N. | 72 | 1.3489 | 7.51 | Spasmodic cough since 6 months. |
| J. U. | 24 | 1.3490 | 7.57 | Spasmodic cough since 3 months. |
| U. F. | 3 | 1.3492 | 7.69 | Spasmodic cough since 40 days. |
| W. F. | 10 | 1.3490 | 7.58 | Spasmodic cough since 21 days. |
| H. A. | 36 | 1.3500 | 8.15 | Spasmodic cough since 7 days. |
| D. A. | 24 | 1.3460 | 5.83 | Nauseous cough 4/18/15. |
| | | 1.3488 | 7.46 | Spasmodic cough 5/10/15. |
| D. C. | 48 | 1.3487 | 7.40 | Spasmodic cough since 10 days. |
| | | 1.3503 | 8.33 | Spasmodic cough since 30 days. |

The researches of Engel, Sandelowsky, Oppenheimer and Reiss, and Bohme all conform to those of Achard, Touraine and Saint-Girons. According to Reiss[4] the fall of protein concentration is due to one of three causes: water retention by the organism; absolute decrease of proteins; water carriage from tissues to the blood stream. In pneumonia and scarlet fever, as shown by Oppenheimer and Reiss, refractometric figures are inversely proportional to weight figures; they have seen that in chlorid crises, weight decrease and refractometric index increase come together. Von Leyden had otherwise observed water retention in fever.

4. Reiss: Ergebn. inn. Mediz. u. Kinderh., 1913, xi.

During fever there is a great protein destruction, which is indicated by an increase of nitrogen output. It is probable that the proteins of the blood stream are also raised, but we do not understand that such

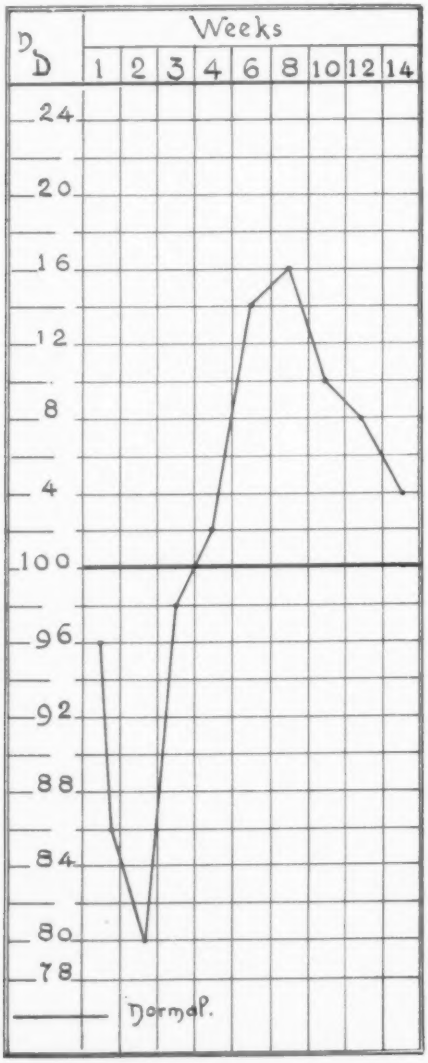


Fig. 3.—Hemorefractometry of whooping cough as compared with the normal.

destruction promotes so great a decrease of protein concentration as that observed. Reiss, however, believes this to be the cause of refractometric index decrease in cases with high fever, weight loss and no chlorid retention.

Water flow from tissues to blood in superwarming was experimentally demonstrated by Sandelowsky, but clinically we observe that fever figures in no way parallel the refractometric figures. In superwarming protein concentration rises forthwith after warming ceases, but in recovery from febrile infections the refractometric index rises little by little. Reiss believes that almost always blood dilution proceeds from water and chlorid retention.

TABLE 4.—Refractometric Index in Latent and Surgical Tuberculosis

| Name | Age, Years | Weight, Gm. | Diagnosis | N/D | Proteins, Per Cent. |
|---|---|---|---|---|---|
| D. B. | ½ | 6,500 | Latent tuberculosis | 1.3460 | 5.83 |
| E. R. | 5/12 | 8,000 | Latent tuberculosis | 1.3477 | 6.81 |
| G. S. | ½ | 6,900 | Latent tuberculosis | 1.3475 | 6.70 |
| U. B. | ¾ | 5,000 | Adenopathy | 1.3448 | 5.13 |
| D. F. | 1 1/12 | 7,200 | Adenopathy | 1.3458 | 5.71 |
| A. A. | 10/12 | 9,800 | Adenopathy | 1.3468 | 6.29 |
| A. S. | 8 | 24,900 | Adenopathy | 1.3482 | 7.11 |
| M. J. | 7 | 16,000 | Adenopathy | 1.3476 | 6.76 |
| C. S. | 7 | 20,000 | Adenopathy | 1.3478 | 6.87 |
| V. R. C. | 11 | 29,000 | Serous pleurisy | 1.3499 | 8.09 |
| U. C. | 6 | 13,500 | Pott's disease with pulmonary tub. | 1.3401 | 6.47 |
| A. D. | 3 | 13,000 | Pott's disease | 1.3510 | 8.73 |
| J. J. | 7 | 22,700 | Coxalgia | 1.3507 | 8.56 |
| A. A. | 9 | 25,000 | Coxalgia | 1.3488 | 7.46 |
| W. D. | 8 | 24,200 | Scrofula | 1.3478 | 6.87 |
| C. S. | 2½ | 6,300 | Pulmonary tubere. | 1.3439 | 4.61 |
| H. T. | 1 7/12 | 5,400 | Pulmonary tubere. | 1.3449 | 5.19 |

It is very interesting to compare the results from observation of infectious diseases and those which have been shown by Widal, Abrami, Brissaud, Benard and Joltrain[5] in anaphylaxis. They observed sudden fall of the refractometric index, and believe these variations to be due to physicochemic alterations of plasmatic colloids.

In order to see if in infancy refractometric figures were the same as in adults, I have examined serumprotein concentration in infants and children at the Children's Hospital Dispensary at Rio de Janeiro, where I was consulting physician.

I have divided this research into two parts: on acute, and on chronic infectious diseases. Concerning acute diseases, I have found

5. Widal, Abrami, Brissaud, Benard and Joltrain: Compt. rend. Soc. de biol., 1914.

in measles a figure identical with that of Achard, Touraine and Saint-Girons, as I show in the observations of Table 2.

The refractometric index is an excellent aid in the differential diagnosis from rubeola. In this last disease protein concentration suffers no variation as I have observed in the following case:

Ismenia V., 7 months old, had a low fever for three days, with an irregular eruption. Koplik —; Theodor +; fever 37.6 C.; n/D = 1.3472. Proteins 6.52 per cent. After four days the value of n/D was the same.

The refractometric index in the course of measles in regard to normal is as shown in Figure 2.

As whooping cough is an apyretic, subacute infection, it was very interesting to ascertain if the Achard, Touraine and Saint-Girons curves were here the same. Researches were made in nauseous and spasmodic periods of the disease and I observed ever a low albuminemy in the first period and a remarkable increase in the last. Table 3 gives a summary of my fourteen observations on this disease.

I have registered in Chart 3 the refractometric index variations from normal in whooping cough.

Refractometry allows here the differential diagnosis from tracheobronchial adenopathy with spasmodic cough. In whooping cough the refractometric index is, during the spasmodic period, very much increased; in tracheobronchial tuberculous adenopathy, on the contrary, it is always decreased.

Concerning chronic diseases, it is very interesting to observe the low refractometric index of tuberculosis in contrast with the high degree of hereditary syphilis. Strauss and Chajes[6] found in eight cases of tuberculosis results permitting of no conclusions.

Engel[7] believes the refractometric index to be almost always normal, and says:

A low refraction coefficient must be considered as a poor prognostic sign, inasmuch as it indicates a high degree of disturbance of nutrition. Particularly noticeable is the successive sinking in such cases, in which a rapid progression leads to an early fatal termination.

Nast,[2] in Salge's clinic, found in tuberculous children the albuminemic coefficient always increased from 0.5 to 3 per cent. I have found a fairly constant decreased refractometric index, which is, in latent and surgical tuberculosis, almost normal, and becoming lower

6. Strauss and Chajes: Ztschr. f. klin. Med., 1910, p. 285.

7. Einen niedrigen Refraktions-koeffizienten müssen wir dagegen als einschlechtes prognostiches Zeichen ansehen, da dieses auf eine hochgradige Störung der Ernährung hinweist. Besonders auffällend ist das successive Sinken in jenen Fällen, in welchen jene rasche Progression in kurzer Zeit zum Exitus führt." Wien. klin. Wchnschr., 1910, p. 1573.

and lower, reaching the minimum in the last periods of the disease, as shown in Table 4.

Concerning syphilis, all our records show an increased albuminemy, as will be seen from Table 5.

TABLE 5.—Refractometric Index in Syphilis

| Name | Age, Months | Weight, Gm. | Diagnosis | N/D | Proteins, Per Cent. |
|---|---|---|---|---|---|
| M. P. | 5 | 6,100 | H. S. | 1.3499 | 8.09 |
| T. G. | 13 | 7,500 | H. S. | 1.3484 | 7.23 |
| A. A. | 10 | 5,800 | H. S. | 1.3461 | 5.88 |
| N. L. | 6 | 6,200 | H. S. | 1.3495 | 7.86 |
| L. S. | 8 | 6,500 | H. S. | 1.3481 | 7.05 |
| E. C. | 7 | 7,500 | Hydrocephalus | 1.3505 | 8.44 |
| J. S. | 10 | 6,900 | H. S. | 1.3510 | 8.73 |
| E. V. | 5 | 5,100 | H. S. | 1.3481 | 7.05 |
| J. C. | 8 | 5,000 | H. S. | 1.3505 | 8.44 |
| J. U. | 11 | 6,600 | H. S. | 1.3485 | 7.28 |
| D. L. | 8 | 6,800 | H. S. | 1.3500 | 8.15 |
| T. S. | 3 | 4,400 | H. S. | 1.3480 | 7.0 |
| D. V. | 6 | 4,600 | H. S. | 1.3480 | 7.0 |
| M. S. | 5 | 6,000 | H. S. | 1.3482 | 7.11 |

In the observations in Table 5 we find an exception in A. A., aged 10 months, with only 5.88 per cent. as the value of the blood-serum-protein coefficient. This infant was atrophic (weighing 5,800 gm.), with alimentary decomposition and signs of tuberculous adenopathy.

## CONCLUSIONS

1. The refractometric index of blood serum in nurslings is lower than that of the adult, and increases slowly from the first month till the age of 13 to 18 months, reaching then a definite value.

2. Achard, Touraine and Saint-Girons' albuminemic curve is constant in acute infectious diseases of infancy and childhood.

3. The spasmodic period of whooping cough produces high albuminemy, which permits the diagnosis from tuberculous tracheobronchial adenopathy.

4. The hemorefractometric coefficient in tuberculosis is generally lower than normal.

5. Syphilis increases remarkably the protein percentage in blood serum.

In conclusion we wish to express our thanks and indebtedness to Dr. Fernandes Figueira, director of the Children's Hospital.

# A STUDY OF THE BLOOD IN TUBERCULOUS MENINGITIS *

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A lack of uniformity in the blood picture in tuberculous meningitis has been frequently noted in the Babies' Hospital during the last three or four years. The variations were so frequent and inexplicable that an effort was made to establish an average figure for the blood count in this condition. For this purpose 169 cases of tuberculous meningitis from the records of the hospital for the last eight years were analyzed. In the course of this work additional facts of interest were obtained, and as they help in some measure to explain the variations in the blood picture of the disease in question, they are worthy of record.

There were 252 counts made on the 169 cases. The average leukocyte count was 20,900 per c.mm. with an average polynucleosis of 72.6 per cent. Only ten, or 4 per cent., of the counts were below 10,000, and twenty, or 8 per cent., were above 30,000. The lowest total count was 5,400, and the highest 70,000—the latter occurring in an uncomplicated case. The polymorphonuclear percentage ranged from 26 to 91. The age of the children varied between 2 months and 4½ years.

It was thought that the variations in the blood picture might be explained by the presence or absence of an added infection, but an attempt to establish this relationship was not successful. For instance, there were four cases showing at some stage of the disease a blood count of 50,000 or over; but none of these cases had any clinical evidence of secondary infection. Three of these came to necropsy where the clinical findings were confirmed.

The correlation of the leukocyte count and the stage of the disease was next undertaken. The result is shown graphically in Chart 1. The broken line indicates the polymorphonuclear percentage and the solid line the total leukocyte count. The upper figures at the bottom of the chart indicate the number of counts made to determine the average polymorphonuclear percentage, and the lower figures the number of counts made to determine the average total leukocyte count. Observations on a number of cases had to be discarded because of the uncertainty of the exact date of onset of the disease. The symptoms that were of most value in fixing the onset were, in order of importance,

* Submitted for publication Dec. 20, 1915.

(1) vomiting, (2) convulsions or twitchings, (3) drowsiness. There were a few counts made in what was judged to be the fifth or sixth week of the disease, but the number was too small to admit of any deduction.

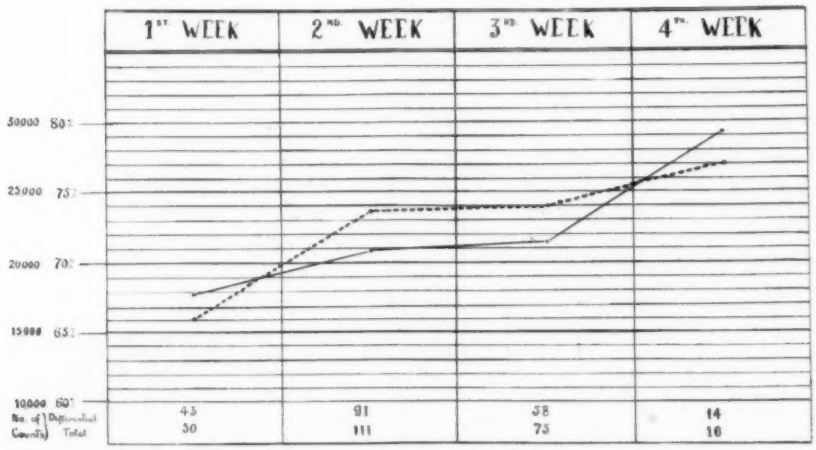


Chart 1.—Showing the gradual increase of the leukocyte count and polymorphonuclear percentage from the first week to the fourth week of the disease.

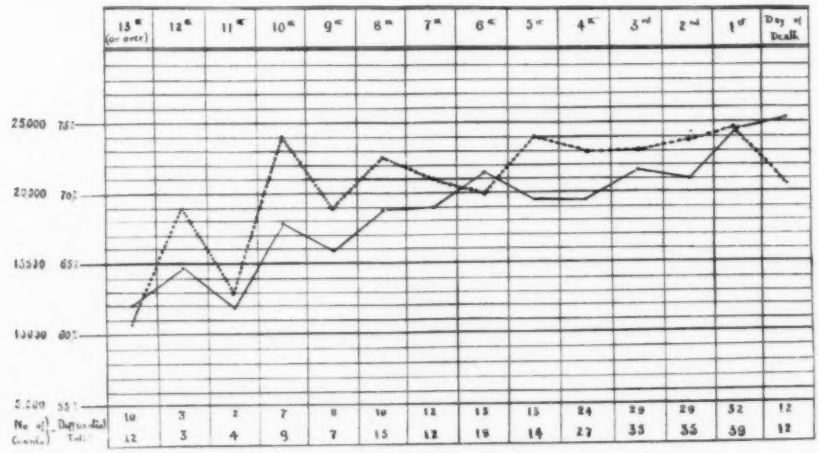


Chart 2.—The average leukocyte count and average polymorphonuclear percentage on the successive days before death, showing the gradual rise as death approaches.

Chart 2 shows the same facts from another point of view. The blood counts on the day of death, the day before death, etc., up to the thirteenth (or over) day before death were grouped together and averaged. The irregularity in the polymorphonuclear percentage curve

in the first four divisions of the chart is no doubt due to the fact that the number of counts was too small to give a very reliable average.

Chart 3 shows the blood picture of a typical case. These figures were kindly lent me by Dr. Charles Hendee Smith.

The observation that an increase in the polymorphonuclear percentage occurs in cases of tuberculosis which are progressing unfavorably is by no means a new one. Many observers, both in this country and abroad, have reached similar conclusions, but have based them on a study of cases of pulmonary tuberculosis. In such patients it is difficult to eliminate the possibility of a secondary infection in the lungs as the determining factor in the production of the increased polynucleosis. In tuberculous meningitis, however, a secondary infection is unusual, and for this reason the foregoing results are of consid-

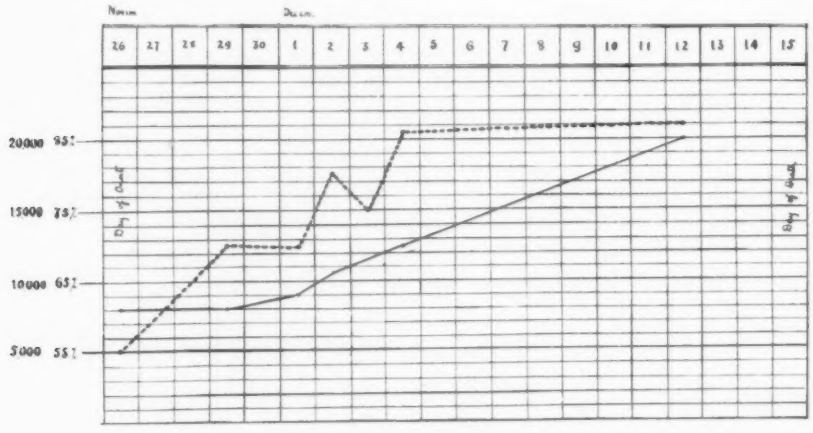


Chart 3.—The blood count in a typical case.

erable confirmatory value. These results differ from those obtained in pulmonary tuberculosis in one respect. In the latter disease, increase in the total leukocyte count has been slight, which indicates that the increase in the percentage of polymorphonuclear cells has taken place at the expense of the mononuclear cells. In tuberculous meningitis, however, as an analysis of the above figures shows, the increase in the polymorphonuclear percentage is accompanied by a parallel increase in the total leukocyte count, so that the total lymphocytes remain practically stationary. This fact makes it more difficult to offer an adequate explanation for the changes in the blood.

The character of the tuberculin skin reaction is to some extent an index of the resistance of the patient to the tuberculous infection. In several cases in this series the skin test was made every second day during the child's stay in the hospital. It was interesting to observe the gradual diminution in the intensity of the reaction as the disease

advanced. It seemed reasonable to expect a definite relationship between the blood picture and the intensity of the tuberculin skin reaction. To demonstrate this, the reactions were divided into four groups, (1) strongly positive (+ + or + + +), (2), positive (+), (3) faintly positive, (4) negative. The blood counts in these cases, made at the same time the skin test was applied, were tabulated and averaged. Chart 4 shows very strikingly the results obtained.

A satisfactory explanation of the changes in the blood picture in tuberculosis has already been advanced. Murphy and Ellis have shown the importance of the lymphocytes in the protection of the body against the tuberculous infection. These cells exert their protective action by virtue of a ferment of lipolytic power, capable of splitting wax and fat

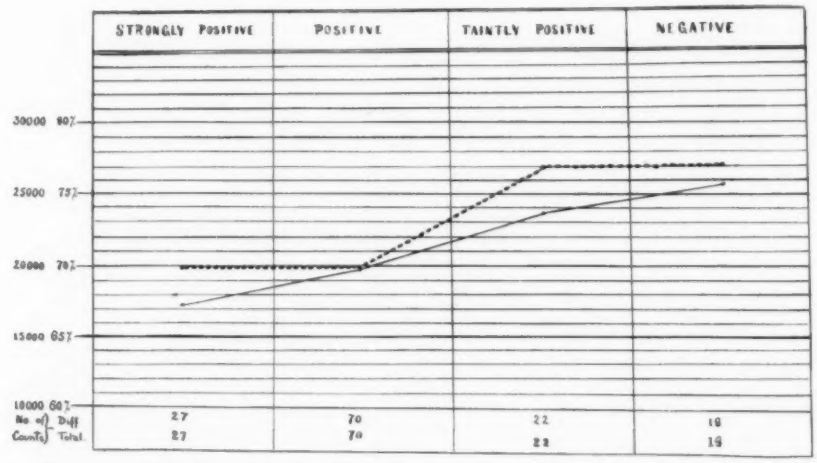


Chart 4.—Showing the relationship between the intensity of the tuberculin skin reaction and the height of the leukocytic count and polymorphonuclear percentage.

into glycerin and fatty acids. It is known that approximately 30 per cent. by weight of the tubercle bacillus is composed of waxy substances, which are broken down by means of this so-called "lympho-lipase," thus rendering the organism susceptible to digestion by the phagocytes. The chronicity of tuberculosis has been thought to be due to the difficulty the defensive mechanism of the body must have to destroy this waxy substance. An increase, then, in the lymphocytes of the blood is usually coincident with improvement in the patient's condition; and conversely, as the patient grows worse, the proportion of polymorphonuclear cells increases at the expense of the lymphocytes.

The figures obtained in this study are of interest from a scientific point of view, but are of less practical value. In the differential diagnosis of obscure cases with meningeal signs, however, it may be of

no little help to know that a blood count of 20,000 to 30,000 with a high polymorphonuclear percentage does not by any means exclude tuberculous meningitis.

## CONCLUSIONS

1. The leukocyte count in tuberculous meningitis is higher than has been heretofore described. The average in this series was 20,900 per c.mm. with 72.6 per cent. polymorphonuclears.

2. The total leukocyte count and the proportion of polymorphonuclear cells vary with the stage of the disease; e.g., both counts increase as the disease advances. There is a relative but not absolute diminution in the mononuclear elements of the blood.

3. There is a definite relationship between the intensity of the tuberculin skin reaction, on the one hand, and the total leukocyte count and polymorphonuclear percentage on the other. Diminution in the former is usually accompanied by an increase in the latter, both being evidences of a failing resistance by the body to the tuberculous infection.

I am indebted to Dr. L. E. Holt for permission to publish this study, and to Dr. Charles Hendee Smith for his kindness in lending me the record of the blood examinations shown in Chart 3.

## REFERENCES

Pavillard, H.: Thèses de médecine de Paris, 1899-1900.
Holmes, A. M.: Med. Rec., New York, 1896, l, 325.
Hultgen, J. F.: Trans. Chicago Path. Soc., 1909, vii, 199.
Swan, J. M.: Jour. Am. Med. Assn., 1904, xlii, 697.
Craig, F. A.: Fourth Annual Report, Henry Phipps Inst., 1908, p. 108.
Solis-Cohen, M., and Strickler, A.: Trans. Nat. Assn. for Study and Prev. Tuberc., 1910, vi, 309.
Solis-Cohen, M., and Strickler, A.: Am. Jour. Med. Sc., 1911, cxlii, 691.
Webb, C. B., Williams, W. W., and Bassinger, A. F.: Trans. Nat. Assn. for Study and Prev. Tuberc., 1910, vi, 281.
Murphy, Jas. B., and Ellis, A. W. M.: Jour. Exper. Med., 1914, xx, 397.
Fiessinger, and Pierre, Louis Marie: Presse méd., 1909, xvii, 406.
Bergel, Salo: Müenchen. med. Wchnschr., 1909, lvi, 64.

# A RAPID METHOD FOR THE DETERMINATION OF FAT IN FECES *

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An important aid in the study of fat digestive disorders of infants and young children is a quantitative estimation of the fat of the stools. Also it is frequently desirable to be able to follow the fat output from day to day as a guide to rational feeding. McClanahan and Moore[1] say:

> We occasionally find cases which seem unable to digest the smallest quantities of fat. They may however have their fat tolerance gradually increased by carefully increasing the fat content of their food as they seem able to digest it. In these cases an increase in the total fat in the stools, especially if persistent and rising, is an indication for the lowering of the fat percentage of the food.

A prime necessity in the study and treatment of such cases is a rapid and easy method for the determination of the feces fat so that necessary changes in the diet may be made promptly.

The methods now in use are unsatisfactory from a clinical standpoint because of the length of time required to make a determination. Standard methods such as the Folin-Wentworth[2] and the Kumagawa and Suto[3] require from twenty-four to thirty hours for a determination and it would be very hard to regulate the daily feedings of infants and young children if we depended on them. A method was sought which could be carried out in a reasonably short time without a notable sacrifice of accuracy.

By means of the nephelometric principle, which has been used by Bloor[4] for the determination of the fat in blood and milk, it has been found possible to make an accurate determination of the fat in the stools in about one hour. The method consists essentially in extracting directly with acidified alcohol and ether, filtering the extract, then precipitating the fat in a watery solution and comparing the cloudy suspension so obtained with that of a similarly prepared standard solution.

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* Submitted for publication Dec. 28, 1915.

* From the Laboratories of Biological Chemistry of the Harvard Medical School.

1. McClanahan and Moore: Am. Jour. Med. Sc., 1915, cxlix, 815.
2. Folin and Wentworth: Jour. Biol. Chem., 1910, vii, 421.
3. Kumagawa and Suto: Biochem. Ztschr., 1908, viii, 212.
4. Bloor: Jour. Biol. Chem., 1914, xvii, 377; Jour. Am. Chem. Soc., 1914, xxxvi, 1300.

*Procedure.*—The whole stool is weighed to centigrams on a previously weighed porcelain plate and then thoroughly mixed with a spatula. A dry and clean 100 c.c. graduated Erlenmeyer flask, together with a 7 cm. filter paper is weighed to milligrams. About 0.5 gm. of the mixed stool is spread out in a thin and even layer on the filter paper, then paper and flask weighed again. The difference between this weight and the original weight of flask and filter paper is the amount of stool taken.

TABLE 1.—DETERMINATION OF FAT CONTENT OF DRIED STOOLS BY FOLIN-WENTWORTH AND NEPHELOMETRIC METHODS

| Dried Stool | Folin-Wentworth, Mg. | Nephelometric, Mg. | Per Cent. Difference |
|---|---|---|---|
| A | 32.8 | 33.6 | +2.3 |
| B | 54.6 | 55.7 | +1.9 |
| C | 57.6 | 58.7 | +1.8 |
| D | 55.4 | 57.5 | +3.6 |
| E | 47.7 | 49.3 | +3.2 |

To the flask are added 60 c.c. to 70 c.c. of a mixture of 3 parts redistilled alcohol and 1 part redistilled ether, and 5 drops of concentrated hydrochloric acid. The filter paper should be entirely immersed in the liquid. The flask is connected with a return condenser and boiled for forty-five minutes then cooled under the tap with shaking, and made up to volume with alcohol-ether, mixed and filtered. The filtrate is usually water clear, but may have a slight tinge of green or brown.

TABLE 2.—COMPARATIVE FAT DETERMINATIONS ON MOIST STOOLS, USING THE KUMAGAWA-SUTO AND NEPHELOMETRIC METHODS

| Moist Stool | Kumagawa-Suto, Per Cent. Fat | Nephelometric, Per Cent. Fat | Per Cent. Difference |
|---|---|---|---|
| A | 10.7 | 10.6 | −1.0 |
| B | 19.1 | 18.7 | −2.0 |
| C | 12.4 | 12.8 | +3.2 |
| D | 9.1 | 9.3 | +2.2 |
| E | 8.3 | 8.9 | +7.2 |
| F | 13.1 | 13.6 | +4.0 |
| G | 15.3 | 15.8 | +3.2 |
| H | 8.7 | 8.4 | −3.4 |

*Determination.*—Five c.c. of the extract are measured from a pipet, with stirring, into a small beaker containing 50 c.c. of distilled water. In the same way 5 c.c. of a standard fat solution (see below) are added from a pipet, with stirring, to another small beaker containing 50 c.c. distilled water. To the standard and to the test solutions are added simultaneously 10 c.c. portions of dilute (1:4) hydrochloric acid with stirring. The solutions are allowed to stand for five minutes, after which they are transferred to the comparison tubes of the nephelometer.

The two tubes are filled to the same height with the solutions and are placed in the nephelometer with the standard tube always on the same side. (If bubbles appear on the walls of the tube it is important that they should be removed by inverting the tube two or three times. At the end of the readings again examine the tubes for bubbles and if they are present the tubes should again be inverted and another reading made.) The moveable jacket of the standard is usually set at 50 mm. (on the original Richards nephelometer)[5] or at 30 mm. in a modified Duboscq colorimeter as described by Bloor.[6] The comparison is made by adjusting the jacket of the test solution until the illumination of the solutions are equal. At least five readings are taken, alternately from below and above and the average taken as the correct reading. The readings are inversely proportional to the values, and calculations are made as in the colorimetric methods.

*Standard Solution.*—The standard solution used is an alcohol ether solution of equal parts of pure stearic and oleic acids, of which 5 c.c. contain about 2 mg. of fat.[7] After trying different proportions of both we found that equal amounts gave readings closest to the results obtained by the Folin-Wentworth and the Kumagawa and Suto methods.

The nephelometric method as above will give consistent results to within 5 per cent. of the results obtained by the older methods, and is therefore adequate for all clinical purposes.

*Results.*—Determinations of the fat content of dried stools by the Folin-Wentworth and the nephelometric methods gave results as shown in Table 1.

Comparative determinations on moist stool using the Kumagawa-Suto and nephelometric methods resulted as shown in Table 2.

501 Linden Street.

5. Richards, T. W., and Wells, R. C.: Am. Chem. Jour., 1904, xxxi, 235.

6. Bloor: Jour. Biol. Chem., 1915, xxii, 145.

7. The standard is most conveniently made by making separate solutions of stearic and oleic acids, each containing about 0.2 gm. in 500 c.c., then mixing smaller volumes as needed. In making the solutions the fatty acid is first dissolved in the ether, then the alcohol is added.

# SIALOLITHIASIS AND SIALODOCHITIS IN CHILDHOOD *

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During the time that observations were made on a recently reported variety of sublingual gland inflammation[1] notes were kept of other affections of the salivary glands seen in children. Three cases of salivary calculus and two of chronic sialodochitis were encountered.[2]

In his classical work on salivary calculus, published in 1855, Thomas de Closmadeuc stated that sialolithiasis did not exist in children; its occurrence in an individual of 20 years was the youngest case recorded at that time. Since then, however, seven cases of salivary calculus in children have been reported.[3] In adding those I have observed, I wish to show that sialolithiasis is not an exceedingly rare condition in children, and to depict its symptomatology in early life.

Chronic inflammation and stenosis of the salivary duct, in the form observed by me in two cases, does not appear to have been described. The affection is of some importance; in both cases the diagnosis of parotid neoplasm had been made and excision proposed. Sialodochitis bears no demonstrable relationship to salivary calculus; it is convenient, however, to discuss both affections in one paper.

For purposes of comparison, the manifestations of sialolithiasis in adults will be considered very briefly. Its frequency in adult life is suggested by some 400 cases that have been described, groups of eight or more often being reported by individual observers. Calculi are encountered most frequently in the excretory ducts; in Wharton's duct in the great majority of instances. The submaxillary gland, likewise, is the most common seat of the gland stones. A distinction between duct and gland concretions is made because it is as yet impossible to determine if duct stones are extruded gland stones, or whether the conditions are separate ones. With the calculus in either situation, inflammatory and sclerotic changes in the gland ensue.

Most observers now agree that sialolithiasis results primarily from bacterial invasion; some, however, presume that swelling of the duct, with retention and inspissation of saliva, is the primary cause, and the

* Submitted for publication Jan. 6, 1916.

1. Neuhof, H.: Am. Jour. Dis. Child., August, 1915, p. 94.

2. With one exception, all were seen and treated in the morning surgical clinic of Mount Sinai Hospital.

3. The reports of only three were found in a careful search of the literature. The remaining four are quoted by various authors, either without reference to their source or with references that were inaccessible or incorrect.

bacterial element secondary. In fact, the etiologic factors are ill understood at present. For example, there is no adequate explanation for the far greater incidence of sialolithiasis in the male sex, or of the preponderence of calculi in the submaxillary duct-gland system, of recurrences after removal, etc.

The symptoms of duct stones are usually more severe and well defined than those of gland stones; calculi in Wharton's duct generally result in the most pronounced manifestations. Wherever the stone is situated there are usually two periods in the symptomatology. The first is characterized by intermittent attacks of pain in the affected side of the mouth, sometimes severe enough to be dignified by the term "salivary colic." The coincident appearance of a sensitive salivary swelling is very characteristic; the mass, tense and firm, is generally in the immediate region of the calculus. Suppuration marks the second stage. Mucopurulent discharge, of varying amounts, escapes from the orifice of the duct, the latter then becoming reddened and swollen. Abscesses about the gland or duct may develop, to burrow externally or ultimately to rupture into the mouth.

A calculus situated near the orifice of a duct is readily palpable through the mucous membrane of the mouth in most instances; in the depths of the duct it is frequently but by no means invariably felt by probing the canal. The diagnosis of a gland stone, on the other hand, is chiefly based on the indirect evidence of the history and the clinical findings; Roentgen-ray examination has revealed calculi in the gland in a few instances.

Many less important symptoms and physical signs have been omitted; enough has been categorically stated to indicate that the diagnosis of salivary calculus would not be difficult if the cardinal manifestations were present regularly. On the contrary, however, the symptoms are often bizarre; acute attacks of sialolithiasis have been mistaken for alveolar periostitis, cervical adenitis, angina Ludovici, etc., and the condition, in a chronic form, has been confused with tuberculosis, gumma, and malignant disease of the salivary gland.

## SIALOLITHIASIS IN CHILDREN

In the reported cases, and in two of the three I have observed, the symptoms were neither as severe as those generally seen in adults nor of as long duration before the patients came under observation. Of my three cases, two were instances of solitary calculus in Wharton's duct, the third had multiple calculi in Stenson's duct. The latter, of unusual interest, will be described first.

Case 1.—A girl, 6½ years old, came under observation in the Mount Sinai Hospital Surgical Clinic in July, 1915 (transferred to me through the courtesy of Dr. S. Cohn). She had had measles and summer diarrhea in infancy.

When she was 2½ years old both ear drums were punctured for an acute inflammation, following grip (?). A swelling of the right side of the face developed soon after; this was termed mumps. The tumefaction, always sensitive and occasionally very painful, has never disappeared since that time. It has been of fairly constant size in summer, decidedly larger and with greater fluctuations in its proportions in the winter. Three or four times in the latter season very acute swelling, with fever, would develop, persist for two or three days, and then slowly recede.

Nine months before the patient was first seen, discharge of pus from the mouth was noted. It has continued ever since. Although usually inconsiderable, thick, and ropy, on a few occasions the discharge has been very profuse and thin. The swelling in the right cheek has been less marked and less painful since the onset of the discharge.

*Examination.*—The child is poorly nourished. General examination is negative. Teeth and buccal mucous membrane are normal. There is a very pronounced tumefaction of the right side of the face, outlining the enlarged parotid gland. On palpation, the gland is very firm and moderately tender; there are a few softer areas in the indurated mass. The orifice of Stenson's duct is very small and reddened, contrasting with the larger, normal orifice on the left side. By pressure on the parotid mass a little saliva mixed with considerable pus is expressed from the duct. The finest lacrymal probe could not be passed into the duct. Roentgen-ray examination was negative. In order to clear up the diagnosis it was determined to split the orifice of the duct and explore its contents.

*Operation.*—Under general anesthesia, the mouth of the duct was widely incised, thick mucopurulent fluid escaping. A good sized probe could then be passed into the duct. At a depth of four cm. a calculus was encountered. After some manipulations the probe could be carried past the calculus to the junction of the duct with the gland. Here a second calculus was felt. Ineffectual attempts were made to extract the stones from the duct.

*Course.*—The parotid swelling began to diminish soon after this operation and the patient felt better. In two weeks the tumefaction had almost disappeared. The discharge of pus steadily diminished, the proportion of saliva increasing with the reduction of the swelling. The calculi, now close together, could easily be felt through the cheek and the mouth, as well as by probing the duct.

One month later the parotid swelling was practically gone, the discharge of pus had ceased, and the child was quite well except for the presence of the calculi in the duct. There was no tendency toward closure of the slit orifice. Another ineffectual effort was made to extract the stones from the duct. Evidently they lay beyond a stricture of the canal, and therefore could not be grasped and removed. An open operation for their removal was refused. When recently seen (October, 1915) the patient was still quite well. The swelling of the parotid was gone. The calculi were easily felt through the cheek.

Case 2.—A boy, 5 years old, came under observation in the surgical clinic of Mt. Sinai Hospital in August, 1911, with a two months' complaint of pain under the tongue and along the floor of the mouth. Pain had been continuous, not severe, and not progressive. Other symptoms had not been noted. There were no previous illnesses other than measles, and nothing in the history of the case to throw any light on the cause of the present affection.

*Examination.*—The general physical examination was negative. The right submaxillary gland was slightly enlarged and sensitive. To the right of the frenum linguae the mucous membrane and submucous tissues of the floor of the mouth were somewhat edematous. In this area a small, firm, rounded mass (the calculus) was indefinitely felt. The orifice of Wharton's duct appeared unchanged, but a fine probe could not be introduced. Saliva could not be expressed from the duct.

*Treatment and Course.*—Under local anesthesia a small incision was made through the duct over the calculus; the latter was then readily extracted. Escape of thick saliva ensued. The stone was somewhat larger than a green pea, egg shaped (the small end pointing to the orifice of the duct), and of slightly irregular surface. On chemical examination (chemical laboratory, Dr. Bookman) it was found to consist entirely of calcium phosphate.

The postoperative course was uneventful. The discharge of saliva continued through the new opening of Wharton's duct. The swelling of the submaxillary gland disappeared. The remainder of the duct was probed on several occasions and found free. About two months after operation the patient disappeared from observation.

Case 3.—A boy, 8 years old, came to the morning surgical clinic of Mt. Sinai Hospital in May, 1912, complaining of a sense of discomfort in the floor of the mouth, of one week's duration. There was no similar previous complaint. Scarlet and diphtheria when 6½ years old. There was no history of any mouth infection or injury.

*Examination.*—The general examination of the robust patient was negative. On the right side of the floor of the mouth, close to the orifice of Wharton's duct, a calculus could be easily felt. The overlying mucous membrane was unchanged. Saliva was not seen escaping from the unaltered orifice of Wharton's duct. The submaxillary gland was not palpable.

*Treatment.*—Under local anesthesia an incision was made over the calculus and was extended through the mouth of the duct. The calculus was extruded from the incision by the dammed-up saliva, which then escaped in considerable quantities. The dilated duct was probed to the gland and was found free. The calculus was slightly smaller than a lima bean, of spheroidal shape, very light and fragile, and of a spongy appearance; unfortunately it was misplaced and was not examined. The patient did not return for further treatment.

The cases of salivary calculus in childhood, whose reports in the literature are accessible, are as follows:

Case 4.[4]—The patient, the youngest on record, was a male infant, 3 weeks old. The mother took the child to the physicians because it nursed with great difficulty. The tongue was found markedly displaced by a large, soft, sublingual swelling, presenting a firmer area in the center. A diagnosis of ranula was made. On pressing the mass, however, the tip of a calculus was seen protruding from the orifice of the duct. After several attempts the calculus was finally removed without an incision. The child remained well. The calculus was the size and shape of a grain of wheat, with one sharply pointed extremity, of yellowish color, and granulated surface. It was composed of phosphate of lime and mucus. The authors believed that it developed in fetal life.

Case 5.[5]—Girl, 12 years old. There was a three months' history of intermittent swelling in the sublingual region. Each recession of the swelling was accompanied by a profuse discharge of saliva and a very small amount of pus. The patient complained of some interference with talking and swallowing. On examination, a calculus was found engaged in Wharton's duct. It could be rolled under the finger. About the calculus there was a soft swelling, the size of a "nut." The duct over the swelling was incised and the calculus removed. Its extraction was followed by the escape of considerable saliva, and the disappearance of the mass. Cultures from the center of the mass were sterile.

Case 6.[6]—A boy, 13 years old. For three years there had been pain in the right side of the mouth during mastication, pain in the right cheek, and inter-

4. Burdel and Cloquet: Comp. rend. Acad. de sc., No. 50, p. 893.
5. Péraire and Gaudier: Bull. Soc. anat. de Paris, 1898.
6. Solé: Presse méd. Belge, 1897, xlix, 113.

mittent swelling of the cheek in cold and damp weather. These symptoms were gradually progressive. On examination, the right cheek was found slightly swollen, the venules of the overlying skin slightly dilated. Palpation revealed a series of nodules in the course of Stenson's duct, more readily felt when the masseters were contracted. By probing, the duct was found abnormally arched. Six calculi were extracted, through an external incision of the duct. They varied in weight from 0.006 to 0.170 gm. The incision in the duct was sutured with catgut; the postoperative course was smooth and there was no leakage of saliva from the wound.

The remaining cases, whose reports are not accessible are: Two cases presumably reported by Schenk, one in a child 7 years old, the other in a child of 12. Wright (in a monograph on the Physiology and Pathology of the Saliva, 1892) is said to have described a salivary calculus in a child 9 years old. Reboul's[7] case occurred in a child 9 (or 15) years old. Beyond the bare statement of the existence of salivary stone in these children the authors referring to them go no further; there are no descriptions of clinical findings or of the results of treatment.

### SIALODOCHITIS IN CHILDREN

Two varieties of chronic sialodochitis have been described. They are affections of adult life; in a very few instances, however, their onset in childhood has been observed. Although they bear no relation to the type of sialodochitis I wish to describe, a short presentation of the clinical pictures may be permitted.

One variety—the sialodochitis fibrinosa of Kussmaul—is characterized by sudden and recurrent attacks of duct obstruction caused by fibrin plugs. This very rare affection is exceedingly chronic, affects Stenson's ducts in most instances, and is generally bilateral. The earliest manifestations of some of these cases date from childhood. The second type is, practically, identical in the recurrence of attacks, in its chronicity, and the almost exclusive, generally bilateral involvement of Stenson's ducts; it is chiefly differentiated by the absence of duct plugs. One case has been observed in childhood:[8] A boy 13 years old suffered from intermittent attacks of pain and parotid swelling for about one year. The duct was dilated, considerable pus escaped from its mouth and the entire course of the duct could be readily probed.

These peculiar, very obscure (and probably constitutional) affections are the only forms of chronic sialodochitis that appear to have been recorded, either in adult life or in childhood. The type I wish to describe is characterized by a cicatricial stenosis of one Stenson's duct (the opposite one being normal), the result of an inflammation of unknown origin, by a firm, nodular enlargement of the corresponding

7. Said to have been reported in the Echo méd des Cévennes, 1902.
8. Noeggerrath: Charité Annalen, 1909, p. 176.

parotid gland, by a tendency to recurrence after slitting the mouth of the duct, but ultimate cure after the stenosis has been permanently overcome.

CASE 1.—A boy 4 years old came under observation in the morning surgical clinic of Mount Sinai Hospital in September, 1913, through the courtesy of Dr. Stephen Greenfield. There was an enlargement in the right parotid region, of five months' duration. The mass had slowly increased in size, pain in and about it had been dull and constant, some loss in weight had been noted. There were no previous illnesses other than measles at the age of 2 years, and pertussis soon after. A diagnosis of sarcoma of the parotid gland had been made and excision advised.

*Physical Examination.*—A poorly nourished pale child. The general examination is negative. The buccal mucous membrane is normal. The left parotid gland and duct reveal no abnormalities. The right parotid gland is enlarged to about three times the normal size; is firm throughout, but with more solid, irregular, pea-sized nodules scattered near the surface and in the depths. The mass is moderately sensitive to pressure. It is fixed to the underlying tissues. There are a few enlarged, tender lymphnodes in the right submaxillary triangle. The parotid duct is felt as a firm, rigid strand in the cheek. The mouth of the duct can be barely distinguished. It makes a tiny pale depression in the buccal mucous membrane; saliva does not escape when the parotid mass is compressed. The finest lacrymal probe cannot be introduced into the orifice of the duct.

The diagnosis of parotid sarcoma would have been concurred in were it not for the duct findings. The parotid swelling itself appeared absolutely typical. Slitting of the duct (in order to explore it) was suggested before sending the case to the wards.

*Treatment and Course.*—General anesthesia. The smallest probe could be passed into the very narrow, stiff-walled duct for about 1 cm. A tight stenosis was then met. With a little manipulation the probe was pushed through a stricture less than 1 cm. long and was then passed into the dilated duct beyond. The cicatricially stenosed part of the duct was slit open from the orifice; the result was a very wide opening leading to the dilated duct. Quantities of thick mucoid saliva escaped. The lining of the dilated part of the duct was then seen to be much thickened, congested, and edematous. A large probe could be introduced to the gland. It described a somewhat irregular course but met no other strictures. The wall of the duct felt very rigid.

Two weeks after operation the parotid tumor had diminished greatly in size and was much softer and more homogeneous in consistency. The only postoperative treatment was pressure made over the gland, at each visit to the clinic (resulting in profuse discharge of saliva from the duct). No visible swelling remained at the end of one month, but the gland was, as yet, firmer and more resistant to touch than the normal. The child disappeared from observation at that time, to return five weeks later with a recurrence of the parotid swelling (to about half the size of the original tumor) and of the stenosis of the duct orifice. The latter was again slit open under general anesthesia; the gland swelling promptly receded. Progressive dilatation of the buccal end of the duct was then practiced for several weeks. Instructions were given for the return of the child twice a month, for the passage of sounds. This was not done, and the patient was not seen again until two and a half months later. The duct mouth was then found partly closed and the parotid gland slightly enlarged. The duct was slit a third time, and there was the same prompt reduction in the size of the parotid. With dilatation by probes for a few weeks the duct has remained patent permanently. When last seen, in July, 1915 (about two years after operation), the patient was well and in excellent general condition. The orifice of the duct was slightly larger than

the normal, could be probed easily; normal-looking saliva escaped; no enlargement of the parotid gland was discernible.

CASE 2.—A girl 8 years old came under my observation in May, 1915, with a swelling in the right parotid region, of four months' duration. The mass increased in size rapidly in the first few weeks, more slowly thereafter. In the first two weeks there was slight fever, and considerable pain in the cheek. The latter was largely controlled by the application of hot poultices. Changes in secretion of saliva were not noted. The physician in charge stated that the parotid swelling was uniform at first, in every way identical with mumps; subsequently, however, the mass became irregularly nodular. There was never any discharge of pus into the mouth. The diagnosis of "mixed tumor" of the parotid had been made, and excision advised. The only suggestive feature in the previous history was that extensive wiring of the teeth to adjust the "bite" had been completed about two months before the onset of the parotid swelling.

The general examination was negative. There was no existing lesion and no evidence of a previous one in the buccal mucous membrane. The left parotid gland and duct were normal. The mucous membrane immediately around the orifice of the right duct was pale and firm to touch. The mouth of the duct was exceedingly small and evidently stenosed. The finest lacrymal probe was introduced with some difficulty. It traversed a very narrow, irregular course for some 2 cm., then suddenly entered the (apparently) dilated duct beyond, and went directly to the gland. The parotid gland was about the size and shape of an English walnut, slightly sensitive, rather firm and resistant to touch, with a number of shotty nodules scattered near the surface and in the depths. The mass was slightly moveable on the underlying tissues. Without the presence of the sound the course of the duct in the cheek could not be felt. (Compare with Case 1.)

The diagnosis of sialodochitis with duct stenosis was made, on the basis of the experiences in the former case. In order to avoid the postoperative recurrences of the stenosis excision of the sclerotic part of the duct was planned.

*Treatment and Course.*—Under local anesthesia the cicatricial tissue around the mouth of the duct was freed. With a probe in the duct as a guide the distal, stenosed end of the canal was isolated and removed with the tissue about the duct orifice. Immediately following the excision there was a profuse flow of glairy saliva mixed with thin watery fluid (cultures sterile). The remaining part of the duct could be probed readily, felt stiff and resistant, and was evidently dilated. A single cat-gut suture approximated the new orifice of the duct to the mucous membrane. Two sutures closed the remainder of the wound to within a short distance of that orifice.

The postoperative course was smooth. Saliva discharged freely from the new duct mouth, the parotid swelling steadily decreased. In three weeks the gland was of almost normal size and much softer in consistency; the nodules were also much softer as well as more indefinite. Two months after operation the gland was barely palpable, the nodules had disappeared, the discharge of saliva was free, the entire course of the duct could be readily probed, and there was no tendency to recurrence of stenosis. When seen recently (November, 1915, six months after operation) the child was quite well; a difference between the parotid glands was not discernible, the new duct orifice remained patent, probing it revealed no abnormalities.

## CONCLUSIONS

1. Sialolithiasis in childhood cannot be termed the exceedingly rare, almost unknown condition it is presumed to be. The manifestations are more clean-cut and evident in children than in adults, the diagnosis

can be made more readily, the surgical treatment is simple and efficacious. The salivary duct should be probed in every instance of enlargement of a salivary gland in a child when a definite cause for the enlargement cannot be ascertained.

2. There is a hitherto undescribed form of sialodochitis of Stenson's duct in children, secondary to inflammation of unknown origin, leading to an enlargement of the parotid gland that can be readily mistaken for sarcoma or mixed tumor. The gland is considerably increased in size, firm, nodular, adherent; the orifice and buccal end of the duct are embedded in stenosing cicatricial tissue. There is a tendency to repeated recurrences of the parotid swelling after slitting the mouth of the duct, but cure follows promptly the excision of the diseased end of the duct.

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# PROGRESS IN PEDIATRICS

## CURRENT PEDIATRIC LITERATURE

### ANATOMY AND PHYSIOLOGY

Carmin Test for Duration of Complete Food Passage in Infants and Children.—A. Hymanson.
Am. Jour. Dis. Child, February, 1916.

Dentition, Human, Problems of.—L. Bolk.
Am. Jour. Anat., January, 1916.

Physiology of Stomach. Effect of Experimental Partial Stenosis of Pylorus on Motility of Empty Stomach.—O. J. Elsesser.
Am. Jour. Physiol., January, 1916.

Physiology of Stomach. Tonus and Contractions of Empty Stomach of Infants with Congenital Pyloric Stenosis, Pylorospasm and Chronic Vomiting (Merycism).—A. J. Carlson and H. Ginsburg.
Am. Jour. Physiol., January, 1916.

### HYGIENE

Infant Life, Conservation of.—E. A. Ayres.
New Jersey Med. Soc. Jour., January 1916.

### BACTERIOLOGY AND PATHOLOGY

Complement Fixation Reactions of Bordet-Gengou Bacillus.—M. P. Olmstead and O. R. Povitzky.
Jour. Med. Research, January, 1916.

Diphtheroid Bacillus of Preisz-Nocard from Equine, Bovine and Ovine Abscesses.—I. C. Hall and R. V. Stone.
Jour. Infect. Dis., February, 1916.

Jaundice, Familial, in the Newly Born.—H. Abels.
Med. Klin., Nov. 28, 1915.

Thymus Transplantation.—Y. Tongu.
Mittiel. a. d. med. Fakultät d. k. Univ. Tokyo, 1915, xiv, No. 2.

### METABOLISM AND NUTRITION

Acidosis.—C. M. Robinson.
Maine Med. Assn. Jour., January, 1916.

Acidosis in Children.
Jour. Am. Med. Assn., Jan. 29, 1916.

Breast-Milk Collector, Improved.—Julius H. Hess.
Jour. Am. Med. Assn., Jan. 22, 1916.

Feeding, Duodenal.—C. R. Jones.
Surg., Gynec. and Obst., February, 1916.

Mother's Milk for Mother's Baby.—W. W. Riha.
New York Med. Jour., Jan. 15, 1916.

Pasteurization of Milk Supplies as a Protection Against Typhoid Fever: Report of an Epidemic.—J. C. Geiger and F. L. Kelly.
Jour. Am. Med. Assn., Jan. 22, 1916.

Tubercle Bacilli, Successful Destruction of, in Milk by Electricity.—J. M Beattie and F. C. Lewis.
Brit. Jour. Tuberc., January, 1916.

## DISEASES OF THE NEW-BORN

Management of the Prematurely Born.—T. Frølich.
Norsk Mag. f. Laegevidensk., January, 1916.

New-Born, Intracranial Bleeding in, Diagnosis of.—H. G. Sloan.
Cleveland Med. Jour., December, 1915.

## ACUTE INFECTIOUS DISEASES

Cerebrospinal Fever.—S. Neave.
Practitioner, London, January, 1916.

Infantile Paralysis.—R. T. Taylor.
New York Med. Jour., Jan. 29, 1916.

Intubation, Forty-Six Cases of, Report of.—E. W. Carpenter.
South Carolina Med. Assn. Jour., January, 1916.

Lobar Pneumonia in Infancy and Childhood.—J. L. Morse.
Boston Med. and Surg. Jour., Feb. 3, 1916.

Meningitis, Acute, Case of, Associated with Acute Suppurative Otitis Media, Both Produced by Streptococcus Mucosus Capsulatus.—R. L. Loughran.
Ann. Otol., Rhinol., and Laryngol., September, 1915.

Meningitis, Case of Pneumonia Complicated by.—A. D. Patillo.
Texas State Jour. Med., December, 1915.

Meningitis, Cerebrospinal, Metastatic Endophthalmitis in Case of.—A. L. Meakley.
Brit. Med. Jour., Jan. 8, 1916.

Meningitis, Cerebrospinal, Treatment of.—W. Osler and Others.
Practitioner, London, January, 1916.

Meningitis, Epidemic Cerebrospinal, Causes and Prevention of.—H. Sutherland.
Practitioner, London, January, 1916.

Meningitis, Epidemic, Mixed Infection with.—H. Silbergleit and K. v. Angerer.
Deutsch. med. Wchnschr., Jan. 6, 1916.

Meningitis, Fulminating, with Atypical Symptoms.—N. Rosenbaum.
Med. Klin., Dec. 26, 1915.

Meningitis, Influenza: Report of Two Cases.—A. Brown.
Canad. Med. Assn. Jour., December, 1915.

Mumps Appears for First Time in Greenland.—A. Bertelsen.
Hygiea, Stockholm, 1915, lxxvii, No. 22.

Otitis Media and Mastoiditis, Case of Followed by Meningitis, Operation, Recovery. After Eight Years, Development of Cerebral Abscess, Operation, Death.—S. J. Kopetzky.
Ann. Otol., Rhinol., and Laryngol., September, 1915.

Paratyphoid B in a Six Months' Infant.—E. Mayerhofer.
Med. Klin., Nov. 28, 1915.

Poliomyelitis, Anterior, Early Management of.—C. B. Francisco.
Missouri State Med. Assn. Jour., January, 1916.

Scarlet Fever.—F. Leech.
Washington Med. Ann., January, 1916.

Scarlet Fever, Epidemic of.—J. Lemche.
Ugesk. f. Laeger, Jan. 6, 1916.

Scarlet Fever, Twenty Years of, in Town of Twenty Thousand Inhabitants.—K. Ekholm.
Finska läk. sällsk. handl., Helsingfors, 1915, lvii, No. 1.

Smallpox and Chickenpox, Etiology and Laboratory Diagnosis of.—J. N. Force.
Jour. Lab. and Clin. Med., January, 1916.

Sprue, Case of, with Necropsy.—E. H. Falconer and A. H. Rowe.
Am. Jour. Trop. Dis. and Prev. Med., January, 1916.

Sprue, Dietetic Treatment of.—B. K. Ashford.
Am. Jour. Trop. Dis. and Prev. Med., January, 1916.

Whooping-Cough.—N. Benstz.
Nederl. Tijdschr. v. Geneesk., Jan. 8, 1916.

Whooping-Cough, Treatment of, with Disease Products (Sputum) of Patients with Pertussis.—R. Kraus.
Wien. klin. Wchnschr., Dec. 23, 1915.

## TUBERCULOSIS AND SYPHILIS

Crippled Tuberculous Children.—H. J. Gauvain.
Brit. Jour. Tuberc., January, 1916.

Lupus, Natural and Artificial Heliotherapy of.—A. Jesionek.
Ztschr. f. Tuberk., December, 1915.

Meningitis, Primary Tuberculous, So-Called, Diagnosis of.—D. W. K. Moody.
Lancet, London, Jan. 1, 1916.

Syphilis, Inherited, and the Wassermann Reaction.—E. Steinert and E. Flusser.
Arch. f. Kinderh., Nov. 2, 1915.

Tuberculosis, Disseminated Miliary.—W. P. Northrup.
Arch. Pediat., December, 1915.

Tuberculosis in Infancy.—C. H. Dunn.
Am. Jour. Dis. Child., February, 1916.

Tuberculous Arthritis, Treatment of.—J. J. Nutt.
Am. Jour. Orthop. Surg., January, 1916.

Tuberculous Meningitis, Ninhydrin Reaction in the Cerebrospinal Fluid in.—E. Nobel.
München. med. Wchnschr., Dec. 28, 1915.

## GASTRO-INTESTINAL SYSTEM

Gastrotomy in Baby Aged Six Months.—C. P. G. Wakeley.
Lancet, London, Jan. 22, 1916.

Hernia, Congenital, Unusually Large Scrotal.—W. B. Jennings.
New York Med. Jour., Jan. 22, 1916.

Hernia, Inguinal, Relapse after Lorthioir's Operation for, in Three Months' Infants.—Bjerrum.
Hygiea, Stockholm, 1915, lxxvii, No. 22.

Poisoning from Canned Vegetable.—A. Baginsky.
Arch. f. Kinderh., Nov. 2, 1915.

Pyloric Obstruction in Infancy.—G. L. McGonigal.
Kansas Med. Soc. Jour., January, 1916.

Pylorospasm in Young Infants.—E. Lövegren and W. Pipping.
Finska Lak. Handl., 1915, lvii, No. 4.

## RESPIRATORY SYSTEM

Foreign Bodies in the Bronchi; Sixteen Cases.—A. Thornval.
Ugesk. f. Laeger, Dec. 23, 1915.

Intrathoracic Lesions in Children, Value of Roentgen Ray in.—R. G. Freeman.
Arch. Pediat., December, 1915.

Pneumonia, Foreign Body.—A. Caillé.
Arch. Pediat., December, 1915.

## BLOOD AND CIRCULATORY SYSTEM

Anemia, Chronic Simple, in Girl of 6, Apparently Cured by Horse Serum Tablets.—A. Buzzo.
Semana med., Oct. 7, 1915.

Bothriocephalus, the Blood Picture in Children with.—G. Becker.
Finska Lak. Handl., 1915, lvii, No. 5.

Differential Counts and Neutrophil Blood Pictures of Natives—Adults and Children—of New Guinea.—A. Breinl and H. Priestley.
Ann. Trop. Med. and Parasit., December, 1915.

Gaucher's Disease; Report of Two Cases in Infants.—J. H. M. Knox, Jr., H. R. Wahl, and H. C. Schmeisser.
Bull. Johns Hopkins Hosp., January, 1916.

Hemophilia, Metabolism Studies in.—M. Kahn.
Am. Jour. Dis. Child., February, 1916.

Strophanthin for Failing Compensation.—A. Baginsky.
Arch. f. Kinderh., Nov. 2, 1915.

## NERVOUS SYSTEM

Amaurotic Family Idiocy, Case of, in One of Twins.—C. Herrman.
Arch. Pediat., December, 1915.

Amyotonia Congenita.—K. H. Krabbe.
Hospitalstid., Jan. 12, 1916.

Convulsions in Children, Atropin for.—F. Rascher.
München. med. Wchnschr., Jan. 4, 1916.

Convulsions of Young Children.
Jour. Am. Med. Assn., Jan. 22, 1916.

Defective Children.—M. H. Kerr.
Public Health Jour., December, 1915.

Delinquents, Defective, under Care of Massachusetts State Board of Insanity, Survey of.—A. W. Stearns.
Am. Jour. Insan., January, 1916.

Epilepsy, Idiopathic, Diagnostic Methods and Pathologic Constants in.—C. A. L. Reed.
Jour. Am. Med. Assn., Jan. 29, 1916.

Epilepsy, Sensory Phenomena in.—E. M. Auer.
Am. Jour. Insanity, January, 1916.

Hydrocephalus, Types of; Their Differentiation and Treatment.—C. H. Frazier.
Am. Jour. Dis. Child., February, 1916.

Macewen's Sign.—H. B. Wilcox.
Arch. Pediat., December, 1915.

Mental Defectives, Experimental and Clinical Studies on.—A. W. Peters and C. D. Blackburn.
Jour. Lab. and Clin. Med., December, 1915.

Tetany, Infantile.—S. Sheill.
Dublin Jour. Med. Sc., January, 1916.

## GENITO-URINARY SYSTEM

Enuresis, Nocturnal, Treatment of.—M. Mendeloff.
West Virginia Med. Jour., February, 1916.

## OSSEOUS SYSTEM

Femur, Separation, Complete, of Head of, in Small Boy; Treatment as for Congenital Dislocation.—A. Broca.
Presse méd., Dec. 9, 1915.

Hip Disease, Quiet (Perthes' Disease).—H. L. Taylor and W. Frieder.
Surg., Gynec. and Obst., February, 1916.

## SKIN AND APPENDAGES

Bullous Keratolysis—Blistering and Peeling of the Skin; Five Cases.—A. Baginsky.
Arch. f. Kinderh., Nov. 2, 1915.

Heliotherapy in Diseases of Skin; Report of Cases.—H. P. Towle.
Jour. Cutan. Dis., December, 1915.

Urticaria in Children.—C. J. Hailperin.
New Jersey Med. Soc. Jour., December, 1915.

## EYE, EAR, NOSE AND THROAT

Ametropia in School Children.—E. L. Russell.
Missouri State Med. Assn Jour., January, 1916.

Audition, Severely Impaired, in Childhood.—L. M. Hubby.
Med. Rec., New York, Dec. 25, 1915.

Gonorrheal Ophthalmia, Treatment of.—H. Gifford.
Southwest Jour. Med. and Surg., January, 1916.

Pneumococcus Sore Throat in Children.—F. P. Gengenbach.
Colorado Med., January, 1916.

Tonsil in Its Relation to Rheumatic Infections.—T. H. Halsted.
New York State Jour. Med., November, 1915.

Tonsil Technic.—R. H. Woods.
Illinois Med. Jour., January, 1916.

Tonsil, Why Remove?—I. D. Kelley, Jr.
Missouri State Med. Assn. Jour., January, 1916.

Tonsillectomy.—J. M. Matthews.
Jour. Am. Med. Assn., Feb. 12, 1916.

Tonsillectomy, Standard.—J. R. Fletcher.
Ann. Otol., Rhinol. and Laryngol., September, 1915.

Tonsils, Diseased, as Factor in Constitutional Diseases, Importance of Recognizing and Appreciating.—V. C. Tisdal.
Oklahoma State Med. Assn. Jour., December, 1915.

## SURGERY AND ORTHOPEDICS

Cleft Palate, Surgical Anatomy of.—J. R. Eastman.
Indiana State Med. Assn. Jour., December, 1915.

Congenital Deformities of Vertebrae and Ribs; Report of Four Cases.—F. G. Hodgson.
Am. Jour. Orthop. Surg., January, 1916.

Crippled Children at Rainbow Hospital, Cleveland, Treatment for.—A. Patton.
Cleveland Med. Jour., October, 1915.

Fracture in Young Infants, Treatment of.—E. Buchheim.
Deutsch. med. Wchnschr., Dec. 9, 1915.

Harelip and Cleft Palate Surgery, Principles of.—J. R. Eastman.
Lancet-Clinic, Nov. 28, 1915.

Hip Joint, Traumatic Dislocation of in a Child.—H. Platt and H. M. von Mengershausen.
Lancet, London, Jan. 8, 1916.

Palate, High Arched, Injurious Influence of, in Children, and Means to Remedy It.—R. Landsberger.
Arch. f. Kinderh., Nov. 2, 1915.

Pott's Disease Treated by Operation: A Report of Six Hundred and Eighty-Two Cases.—W. E. Wolcott.
Jour. Am. Med. Assn., Jan. 8, 1916.

Tarsectomy for Relapsed Club-Foot.—A. G. Cook.
Am. Jour. Orthop. Surg., January, 1916.

Tendon Fixation in Infantile Paralysis; Review of One Hundred and Fifty Operations.—W. E. Gallie.
Am. Jour. Orthop. Surg., January, 1916.

## THERAPEUTICS

Potassium Iodid in Acute Disease of the Respiratory and Cardiovascular Apparatus.—A. Baginsky.
Arch. f. Kinderh., Nov. 2, 1915.